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In Memoriam

REGINALD CHEYNE ELMSLIE, O.B.E.
(1878-1940)

IN losing Elmslie, who died on July 24 at the age of 62, we have to deplore the loss of one who had made a great mark in the special branch of surgery which he had followed for nearly all his professional life.

He was the son of Captain J. A. Elmslie, R.N.R., and his mother was descended from one of the Founders of the Royal College of Surgeons of Edinburgh. He was educated at Brighton Grammar School and entered St. Bartholomew's Hospital at the early age of 17.

He had a distinguished career as a student, acting finally as House Surgeon to Sir Henry Butlin, later obtaining the Gold Medal of the London M.S. and the F.R.C.S. He then joined the teaching staff as Demonstrator in Pathology, a post which he held for some years. This undoubtedly gave a definite turn to his outlook on his own specialty, because his best remembered papers, published in this JOURNAL—of which for many years he was on the Editorial Committee—both dealt with the pathological changes of cysts and fibrocystic diseases of the bones (vol. ii, xx). In 1912 he became Orthopædic Surgeon to St. Bartholomew's Hospital; this was a great innovation because hitherto orthopædics had been regarded as a very minor department and had been in charge of one of the Assistant Surgeons who gave it one or two afternoons a week. At that time the purely orthopædic surgeon was almost unknown in any of the London hospitals.

At the outbreak of the last war he became associated with Sir Robert Jones, and under that great pioneer took an active share in the organization of the Military Orthopædic Hospital at Shepherd's Bush, and Queen Mary Hospital, Roehampton.

He was one of the founders of the British Orthopædic Association, being one of the small group, a dozen or so, who met Robert Osgood of Boston, with Sir Robert Jones and Platt, of Manchester, at an informal dinner which inaugurated that scientific body of which he was later President and always a very energetic member.

He was one of the leading spirits in the reorganization of the Royal National Orthopædic Hospital, and he took a great interest in the extension of that institution to its country branch at Stanmore.

He was elected on the Council of the Royal College of Surgeons and was serving his second term of office.

Elmslie had already gained a notable distinction in winning the Jacksonian Prize at the College for his Essay on The Pathology and Treatment of Deformities of the Long Bones. He led a very busy life, never refusing to undertake official

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work in which his experience would be of great value. He helped to obtain the Charter of the Society of Massage and Medical Gymnastics and was Chairman of the Executive Committee of the Central Council for the Care of Cripples.

By nature he was shy, retiring, and gentle, so that he made many friends and few enemies. The care and devotion which he spent in his ward work and his teaching gave him a large following of students and disciples, who, scattered chiefly in the South of England, will for a long time keep green his memory. He leaves a wife and son of 20.

ON CAROTID TUMOURS

By GORDON GORDON-TAYLOR, Surgeon Rear-Admiral

Carotid tumours are affirmed by some authors to be rare, by others (Talman,[1] Matyas,[2] and others) as the rarest of all tumours of the neck. Although this latter claim may perhaps be challenged by other neoplasms, for example tumours of the parathyroid, the infrequency of carotid tumours is such that many of the contributions to the literature of the subject are based upon a solitary case which has chanced to come under the notice of the reporter. Moreover there is a certain monotony about most of the contributions, for each solemnly reiterates the statement that "the number of carotid tumours reported is steadily increasing"; inasmuch as the augmentation in number is effected by single additions to the collection, the pyramid of our knowledge of these tumours has been only slowly erected since the first case was reported by Scudder in 1903.

The material of the Mayo Clinic invariably provides golden opportunities for its associates, and in 1931 Rankin[3] from that Clinic was able to report no fewer than 12 carotid tumours in 11 patients of his own, one of these having a bilateral growth of the gland. In this country Hamilton Stewart,[4] of Bradford, reported from St. Thomas's Hospital 4 cases that had been operated upon by several surgeons of that famous institution (among them Max Page and the late Percy Sargent). Of others, Talman, of Leningrad, has dealt with 2 cases, and the writer has encountered 5 cases of these tumours.

The main purpose of this communication is to plead once more for caution before undertaking a resection of the carotid bifurcation for a carotid tumour, and to impress upon operators that even when the arteries appear almost imbedded in the growth, meticulous care and painstaking dissection may sometimes reveal a 'white line', whereby the continuity of the main arterial vessels may remain undisturbed, or at least structurally inviolate.

The relatively advanced stage at which most of these cases come under the notice of a skilled observer imposes upon the surgeon the difficult decision between the maintenance of an intransigent respect for the integrity of the carotid bifurcation, and on the other hand, the prospect of an anxious, tedious piece of technique which awaits his execution, if caution wisely prevails. Earlier operation would furnish a simple and safe exercise untrammelled by the thought of any surgical Scylla or Charybdis.

Age.—It is said that these tumours may arise at any age from 7 to 73 years, but Wossnessensk in 1923 operated upon a child of only 6 months for increasing dyspnœa. The growth in this case was said to be a perithelioma, and despite the fact that the tumour could not be radically removed because of fixation to the great vessels, the child was free from recurrence five years later.

My own patients have been almost exclusively in the early part of the seventh decade, although one was only 52, and a glance at a number of cases selected from the literature at random seems to show that many or most of these cases are encountered by operators at an age of about 60 years.

Sex.—Males are said to be more frequently affected than females in the ratio of 3 : 2. Of my own cases 4 were men and 1 was a woman.

Injury.—In one case reported by Joyce and Diack[5] the origin of the tumour was attributed by the patient to an injury. While scuffling with a "friend", she received a moderately hard blow on the left side of the neck; there was no immediate discomfort, but in from 24 to 48 hours a hard swelling appeared, and yet she kept this for 27 years before coming for surgical relief at 60 years of age!

Tumour of Aberrant Carotid Gland.—Rössing[6] at an autopsy found a tumour at a lower level than the carotid bifurcation which had the histological characters of a carotid tumour.

Bilateral Tumours of the Carotid Gland.—Such cases are naturally most exceptional, but Rankin[3] encountered such a patient in whom the tumour on the left side of the neck was malignant and that on the right side benign. Enderlen[7] resected the carotid bifurcation for a tumour on the right side, effecting an anastomosis between the common and internal carotid arteries; fifteen days later he removed a carotid tumour on the left side, but succeeded in conserving the integrity of the arterial trunk. Marangos,[8] of Athens, had also a bilateral tumour, as likewise Lund,[9] although in the case of the Boston surgeon the second tumour appeared on the opposite side of the neck twenty-nine years after the removal of the first.

Malignancy.—Rankin in his 11 patients is said to have found that 6 were malignant and 5 benign, but this is a very low percentage of benign tumours when compared with the figures of other authors. Some affirm that more than 80 per cent are simple, and that in only 8·9 per cent does definite recurrence take place after operation. Of Rankin's 11 patients 9 survived operation, and 7 remained free from recurrence. In such tumours as are definitely malignant cells may be found infiltrating the vessel walls and invading the capsule.

Lymphatic Glands.—Kopfstein, Keen, and Kretschmer have reported cases where the regional lymphatic glands have been infected.

Metastases.—Hastings Gilford[10] reported a case in which there were metastases in the liver, and in Mönckberg's[11] patient there developed a metastasis in the ovary four years after the initial operation.

Concomitant Pathology.—One carotid tumour was found by Roux-Berger and Tailhefer[12] during the progress of a "block-dissection" of the neck in a case of buccal cancer.

Clinical History and Signs.—In many instances a history extending over years may be obtained, and in the patients of fourteen operators the average time elapsing before surgical advice was sought was about twelve years; in my own few personal cases the history rarely dated more than two years previously. Occasionally the tumour may be present for many years, and may then suddenly take on rapid growth.

Size of Tumour.—Except in the fatal case where the tumour was considerably larger, all my personal cases have been of almost similar size and have measured about 3 in. to 3½ in. in the vertical and transverse diameters. The literature describes tumours as having the dimensions of a hen's egg (de Oliveira[12]), an apricot (Miginiac[13]), an apple (Trinchera[14]), etc. In O'Shaughnessy's[15] case of a male Sudanese the vertical axis of the tumour amounted to 15 cm., and in

L. Schmidt's[16] patient, the tumour measured 12 cm. from its upper to lower extremity by 8 cm. by 5 cm.

The *long axis* is usually said to be vertical, but in my own cases this was by no means the case; the tumours were of a more globular character, the diameter being about 7·5 cm. to 9 cm. The tumour is characteristically painless, and is never tender; but pain referred to the cervical plexus has been recorded by Stanford Cade.[17] On very rare occasions pain has been complained of locally (de Oliveira[12]); a tingling sensation has been mentioned.

Mobility.—The tumour is *not* movable vertically, but can be moved slightly from side to side.

Pulsation is usually communicated, but was expansile in Joyce and Diack's patient; this type of pulsation and the bruit were probably due to the extreme vascularity of the mass and the large cavernous blood spaces.

A systolic murmur is occasionally to be heard over the tumour, and may be transmitted a short distance above the mass. In some patients dyspnœa has been a symptom sometimes induced only on effort (L. Schmidt[16]).

Rarely patients have attacks of syncope; these may be spontaneous (Cassidy,[18] Knighton[19]); the former's patient had six attacks which were cured by the removal of the tumour that had been present for four years. Attacks may be evoked by such apparently trivial causes as a collar round the neck, or even by movements of the head and neck.

Dyspnœa and syncope may be induced by digital compression of some carotid tumours.

The visceral effects of vagal stimulation are not usually obtrusive, but Coke and Dunlop[20] record a case of very marked pre-operative constipation requiring the use of purgatives and enemata which was at once and permanently relieved by the removal of a tumour of a carotid gland implicating the adjacent nerves. Hoarseness and cough may be produced by pressure on the recurrent laryngeal nerve.

Dysphagia has been reported.

Radiosensitivity of Carotid Body Tumours.—Hartmann[22] speaks of these tumours as being radiosensitive, and Pearce Gould[23] employed X rays in a symtomless case of his own, where a tumour had been present for thirteen years. The one case in which it was employed by me and which proved completely radio-resistant was found microscopically to be a neurinoma (a tumour of the sheath of Schwann). Pearce Gould's case responded well to X rays, and is alive and well six years later.

REPORTS OF PERSONAL CASES

Case 1.—The patient was a woman of 63 who had noticed a lump on the right side of her neck for five years; this had grown rapidly in the last few months, and now the clinical aspect of the patient closely resembled one of the 'potato tumours' of the neck described by Jonathan Hutchinson[21] in 1888. The tumour presented markedly at the anterior and posterior borders of the sternomastoid, and a well-marked groove separated the tumour from the lower jaw and parotid region (*Fig.* 79).

The fixation and the connexions of the tumour compelled an ablation of the carotid bifurcation with the neoplasm. A hemiplegia developed the next day and death ensued.

The tumour was said to have been a "malignant endothelioma of the carotid gland".

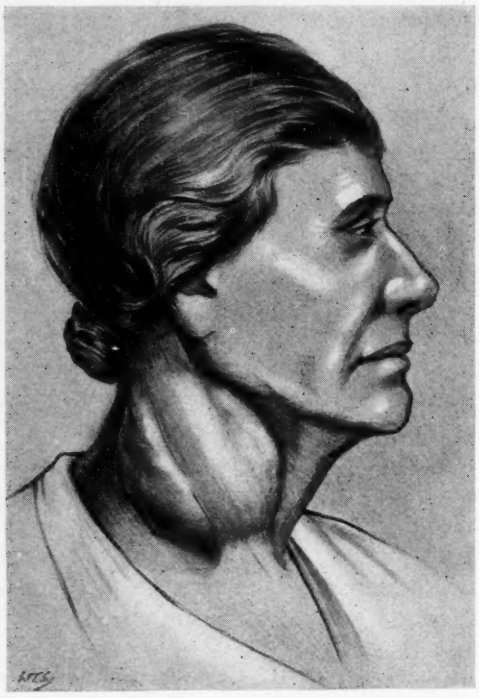


FIG. 79.—*Case* 1. Female patient with 'potato tumour' of right side of neck. The bulging of the tumour at the anterior and posterior borders of the sternomastoid is well shown, and the groove between the upper border of the tumour and the angle of the jaw and parotid. Hemiplegia and death followed resection of the carotid bifurcation.

Case 2.—L. F., aged 54, "non-Aryan" descent. A swelling on the left side of the neck was first noticed in 1925, and at first appeared to have some relation to a dental abscess. The lump persisting, he fell into the hands of a throat surgeon, and his tonsils were naturally enough removed; no change took place in the cervical tumour in consequence of this surgical assault upon the throat. The Wassermann reaction proved negative, and although the mass was single, the possibility of Hodgkin's disease was considered. The mass meantime slowly increased in size, and the patient lost weight. Examination for a primary nasopharyngeal malignant focus proved negative.

OPERATION (March, 1927).—The condition was considered to be probably malignant, and the prospect of a radical removal seemed most hopeful if the ordinary technique of a Crile block-dissection of the neck were followed. After the sternomastoid and internal jugular vein had been divided at their lower end and dissected upwards, the tumour was found to be situated at the bifurcation of the common carotid artery. The internal jugular vein was quite independent of the tumour, but the vagus was so incorporated in the mass that a considerable segment of the nerve had to be resected; the cervical sympathetic trunk and many of its branches were also inextricably implicated. The anxieties associated with interference with the carotid trunk were prominently to the fore, partly because of the disaster in a previous case, partly by reason of the aftermath of carotid artery injury and ligation in the War of 1914–8; every effort was made to remove the tumour without serious interference with the carotid artery.

The growth was situated on the deep aspect of the carotid bifurcation, and *Fig.* 80, A, shows the grooves made by the three arteries in the tumour. Careful dissection enabled the tumour to be removed, but the adventitia of the arterial wall had to be taken away with the tumour.

PROGRESS.—Convalescence was uneventful, although of course paralysis of the left vocal cord resulted from the division of the vagus, and enophthalmos and slight drooping of the eyelid followed the resection of the cervical sympathetic. No aneurysmal dilatation has

followed the stripping away of the adventitia of the arterial wall, and the patient is alive and well 13½ years after the operation.

THE SPECIMEN.—*Macroscopically* (*Fig.* 80, A, B): The tumour is of soft consistence, with a well-defined capsule. The cut surface has a spongy appearance produced by the

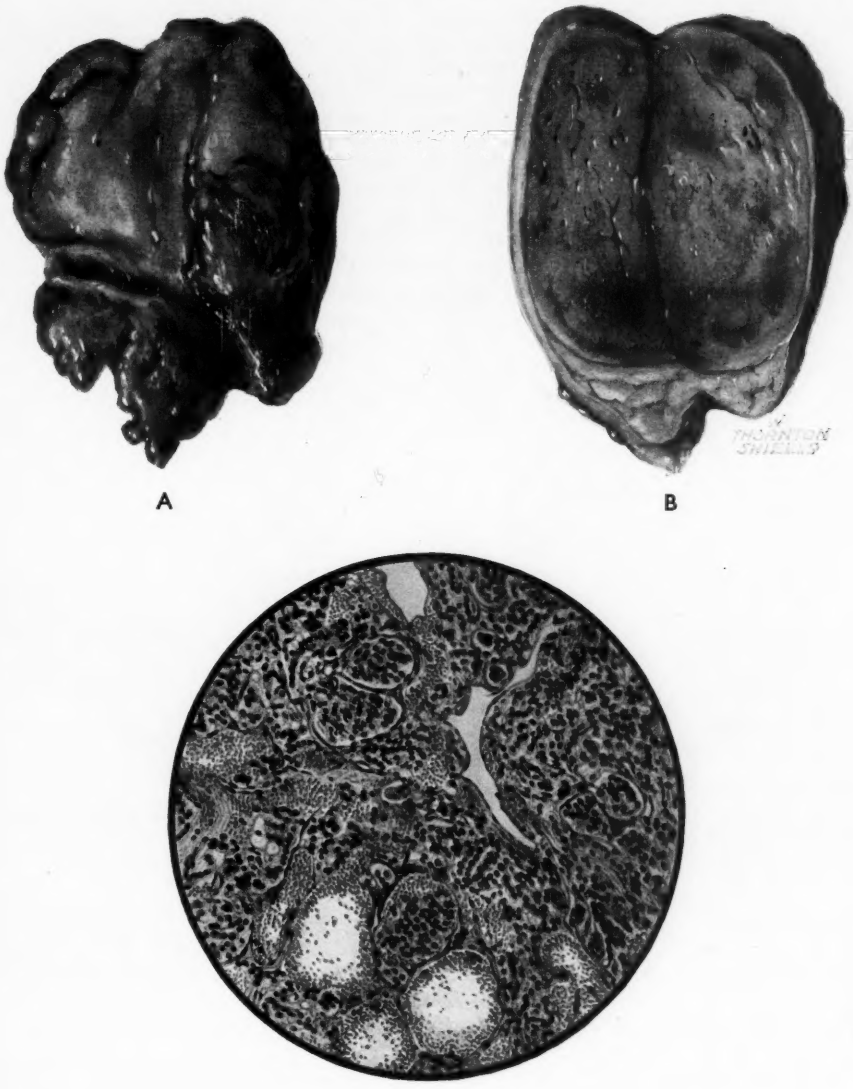


FIG. 80.—*Case* 2. Left carotid gland tumour. A, Showing well-marked grooving of superficial surface of tumour by carotid bifurcation. B, Typical appearance of carotid tumour on section, showing spongy appearance produced by numerous vascular slits and channels. In parts the surface is pale in colour; in other parts there are deep-red patches resembling cavernous angiomata. C, Microscopical appearance.

presence of numerous small vascular slits and channels. In places the surface is pale in colour, in others there are deep red vascular patches resembling cavernous angiomata in appearance.

Microscopically (*Fig.* 80, C): The bulk of the tumour is made up of cells with pale, reticular nuclei. Throughout the greater part of the section the tumour cells are arranged

in a number of closely packed, rounded masses somewhat resembling glomeruli in appearance, but measuring on the average only about half the diameter of a human glomerulus. Each of the small masses of cells is demarcated by a single or double layer of flattened cells. These flattened cells evidently represent a vascular endothelium, since in some cases they form the walls of a vascular space surrounding one of the cell masses. Another striking feature of the tumour consists in the presence of numerous thin-walled vascular channels, which in places form collections of large spaces separated from one another by endothelium only, and having the structure of a cavernous angioma.

Histological examination of several lymphatic glands from the neighbourhood of the tumour failed to reveal any secondary deposits of growth.

Case 3.—P. P., male, 63 years of age, had known of a tumour on the right side of the neck for nearly two years. Growth had been slow; there was no sign of any primary new growth of pharynx or buccal cavity. There was slight movement of the tumour laterally, but no mobility in the vertical direction. There was communicated, but no expansile, pulsation in the tumour, which was approximately the size of a peach. The possibility of a carotid body tumour was discussed before operation.

FIG. 81.—*Case* 3. The internal jugular vein is involved in the tumour as well as the vagus and the sympathetic. The carotid grooving is well shown.

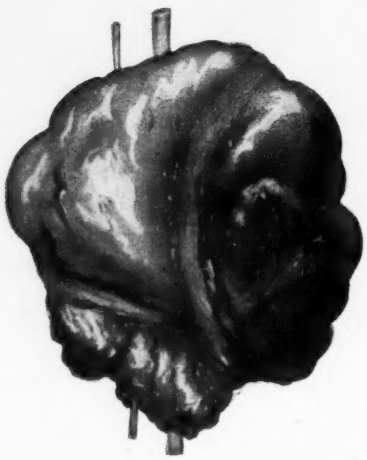


FIG. 82.—*Case* 4. The internal jugular vein was not involved, but the vagus and the cervical sympathetic were implicated as in *Case* 3 (*Fig.* 81). The carotid grooving is well shown.

OPERATION.—At operation, a tumour closely approximating in size to those depicted in *Figs.* 80 and 82 was found; in this case not only were the vagus and the sympathetic involved, but the internal jugular vein was also implicated (*Fig.* 81) and removed in the mass ablated.

Careful dissection enabled the tumour to be removed without interference with the continuity of the carotid trunk.

The patient remains well 8½ years later. The tumour was reported as an "endothelioma of the carotid body".

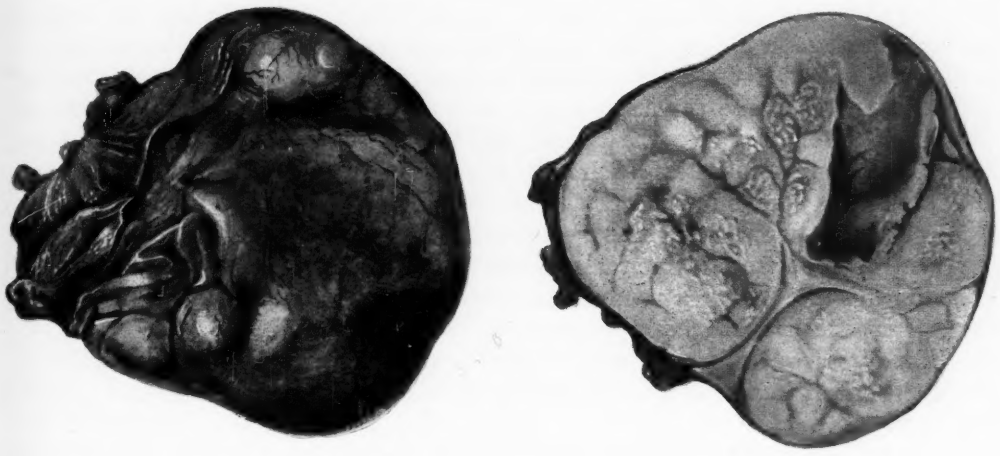
Case 4.—L. P., a male, aged 61 years, had had a lump on the right side of the neck for two years. The size of the tumour closely approximated to that of the former case, and apart from any surgical attentions on the part of otolaryngologists the history was very similar. The possibility of a carotid body tumour was adumbrated before operation.

OPERATION.—The surgery of the tumour was almost precisely similar to that of the previous case. The vagus and cervical sympathetic were alike involved in the tumour and required resection. The internal jugular vein was free. Removal of the tumour was completed by dissection off the carotid bifurcation, with stripping off of the external arterial coat. (*Fig.* 82.)

Recovery was uneventful. The pathological report was a "carotid gland tumour".

Case 5.—E. P., a male, 27 years of age, was admitted into Middlesex Hospital from Kettering with a large tumour of the right side of the neck, which had been present for about nine months. The tumour bulged markedly towards the pharynx. No blood changes were present. The position of the tumour, its characters, and its resemblance to previous tumours of the kind suggested the diagnosis of a carotid tumour with some measure of assurance.

I was, however, overpersuaded to give radiotherapy a trial, but the tumour proved to be completely radioresistant; finally recourse was made to surgery. At operation, the size and relations of the tumour were precisely those of previous carotid tumours encountered



A B

FIG. 83.—*Case* 5. A, Neurinoma removed from the vicinity of the carotid bifurcation of the right side. Throughout the operation the tumour was thought to be an ordinary carotid tumour, and it was only on section of the tumour after its removal that the naked-eye appearances were seen to differ from those which characterize a typical carotid gland tumour (cf. *Fig.* 80, B). The marked grooving of the tumour by the carotid vessels seen in *Figs.* 80–82 is not apparent. B, Naked-eye appearance of the tumour on section.

(*Fig.* 83, A), and no doubt as to the diagnosis was felt until the tumour was cut across at the close of the operation. The macroscopical appearances on section were not those of previous carotid tumours (*Fig.* 83, B); and even on the outer aspect of the tumour there were no grooves due to the carotid vessels, such as had been a noticeable feature of the other specimens.

Microscopically, the tumour proved to be a neurinoma, probably connected with some branch of the vagus or glossopharyngeal supplying the carotid gland.

The man made a good recovery from the operation.

OPERATIVE SURGERY

The anxieties of the operation centre around the possibility of removal of the tumour without resection of the carotid bifurcation or serious interference with the main arterial trunk. Most of the older surgeons plead for caution before undertaking so drastic a step as arterial resection, pointing out that most of the tumours are benign and that surgical resection of the carotid fork is associated with a heavy operative mortality and some prospect of a crippling cerebral aftermath. The operative mortality according to Hartmann,[22] Lenormant, Keen, Bevan,[24] Rankin,[3] and others, approximates to 30 per cent, and is increased by the age of the patient.

Apparently the prognosis of interference with the carotid trunk is rendered safer by preliminary light ligation of the common carotid artery, or even by preoperative compression of the carotid trunk. The former expedient and a two-stage

operation have been successfully employed by Halsted, Enderlen, Jordan, Semler, Perthes, Keynes, and others. Digital compression may be employed from half an hour to an hour and a half twice daily (Talman, Matyas, etc.).

The experience of the last Great War taught British surgeons the value of venous ligation where arterial damage has been done; the ligation of the internal jugular vein will probably not be required as a specific manœuvre, for the vein will doubtless have been resected for reasons of pathology.

Others view interference with the arterial trunk more lightheartedly. Nicolini[25] resected the three great carotid arteries successfully on seven occasions, usually during an operation for malignant glands secondary to cancer of the buccal cavity. L. Bérard and Dargent[26] write enthusiastically of carotid resection in cases of advanced cancer of the cervical glands. A number of single successful operations for carotid tumours where the arterial bifurcation has been resected have been reported by various writers (Schmidt, Oliveira, Talman, Trinchera, Enderlen, O'Shaughnessy, and others).

Enderlen restored the continuity of the arterial trunk by means of an anastomosis between the common and internal carotid artery, stating that he would not have hesitated to make use of a venous graft, had it proved impossible to approximate the arterial ends, as had been suggested by Rehn and Hoffman.

The employment of heparin and the work of Murray, of Toronto, may achieve results in expert hands which will allay the anxieties of those who regard carotid resection with alarm.

Operative mortality or hemiplegia has even followed less serious interference with the arterial tree than resection of the bifurcation. Soupault[27] tied the external carotid at its very commencement, and the patient developed a hemiplegia; Matyas had to ligate the internal carotid, and a hemiplegia ensued.

Lichtenhauer[28] performed a biopsy and, after determining the benignity of the tumour, he investigated the carotid tree by means of arteriography. Finding no vascular obstruction, he refused to perform a resection on the girl of 23!

It may not be inappropriate to quote Bevan's description of an operation for a carotid tumour as an example of judgement and restraint: "As we stood in the operating room with the patient on the table with the tumour and the carotid vessels exposed, these facts flashed through my mind. The patient was under local anæsthesia, so that without any hardship to him one of the assistants went to the library and brought up a volume of Keen's *Surgery* containing the article on tumours of the carotid body, which one of the assistants read to the operating group.

Although it was a great temptation with the complete dissection which had been made to remove this tumour, the Golden Rule was applied. Ask yourself what you would have done with your knowledge as a surgeon under these circumstances, if you were the patient, and you will recognize the fact at once that you would not want your carotid vessels ligated and take a chance of 30 per cent mortality from the operation with the added chance of hemiplegia, even though you survived it."

Although the risk of cerebral complications following carotid occlusion increases with the age of the patient, J. E. H. Roberts[29] with characteristic frankness and honesty records the case of a girl of 23 years in whom he resected the carotid bifurcation with a carotid tumour and who developed a hemiplegia the next day. Recovery ultimately took place.

Operation Findings.—*Figs.* 80–83 illustrate carotid tumours of remarkably similar size; in the fatal case shown in *Fig.* 79 the new growth was rather larger and had grown rapidly. In *Figs.* 80 and 83 the vagus nerve is not demonstrated, although in fact it was hopelessly incorporated in the tumour and had to be sacrificed in every one of my five cases. In each case the tumour had also involved the cervical sympathetic and had developed adhesions to the prevertebral fascia.

The grooves on the superficial aspect of the tumour produced by the carotid bifurcation and the arteries which arise therefrom are well shown in *Figs.* 80–82, but they are not visible in *Fig.* 83, although at the time of operation it appeared to have precisely the same anatomical relations and connexions as the other tumours. It proved to be a neurinoma!

In *Cases* 2–5 the tumour lay on the deep aspect of the carotid fork, and in *Case* 5 bulged markedly towards the pharynx.

In *Case* 1 the carotid bifurcation was hidden in the midst of the tumour and had to be resected, with disastrous results. This case recalls the 'potato tumour' to which Jonathan Hutchinson drew attention in 1888, describing the characteristic manner in which it arises under the sternomastoid and pushes its way both forwards and backwards to assume the shape of a huge kidney potato. Lymphatic glands are not implicated, but the tumour grows rapidly, and usually causes death in from twelve to eighteen months. He regarded these tumours as endotheliomata of the carotid body.

In some tumours the external carotid artery alone is surrounded by the tumour and requires sacrifice, as in Shattock's[30] case. It is indicative of the lowly malignancy of these tumours that whereas the tumour in each of these personal cases implicated the vagus and sympathetic and acquired adhesions to the prevertebral fascia, and although in *Case* 3 the internal jugular vein was incorporated in the mass, nevertheless these cases are alive and well from $8\frac{1}{2}$ to $13\frac{1}{2}$ years after removal of the tumour.

Miginiac, of Toulouse, drew attention to the possibility of syncope during the operation. In his patient, in addition to syncope, there were cyanosis, pupillary dilatation, and apparent death. The attack came on at a stage in the operation when he was making traction on the upper pole of the tumour, and perhaps pinching or pulling the nerve of Hering. This patient fortunately was saved.

In one personal case manipulation of the tumour was said to produce very slight syncope; the vagus was intimately incorporated in the tumour, and when this nerve had been sectioned in the course of the operation all disturbing signs disappeared. The injection of novocain into the tissues of the upper pole of the tumour, if a practicable procedure, or the injection of novocain into the vagus, might prove of value as a prophylactic measure.

Of far less importance in the operative prognosis is the implication of adjacent nerves, such as the vagus, hypoglossal, and sympathetic. Some have said that a consecutive bronchopneumonia with a mortality of 20 per cent follows many cases of vagus section, but this must surely be an exaggeration. It is of course obvious that a laryngeal palsy will complicate vagus section, but pneumonia has not followed any of my own cases, in each of which the vagus has had to be sacrificed.

Interference with the sympathetic will lead to pupil and eyelid changes.

CONCLUSIONS

1. A plea is made for early treatment.

2. The attainment of complete accuracy of diagnosis, despite the increasing number of tumours reported in the literature, must be extremely doubtful, perhaps impossible. The case of the neurinoma which simulated a carotid tumour in every respect at operation reveals the futility of any vaunted pre-operative diagnosis of a carotid tumour.

3. There are grave anxieties attaching to ligature of the common carotid artery or resection of the bifurcation. These anxieties increase with the age of the patient.

4. The part played by the afferent nerves from the carotid sinus in the regulation of the blood-pressure may possibly be of physiological interest, but to the operating surgeon is of little significance and a mere academic chimera and phantasy, which he may disregard light-heartedly.

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CONCERNING THE ORIGIN AND NATURE OF CERTAIN MALFORMATIONS OF THE FACE, HEAD, AND FOOT

By Sir ARTHUR KEITH, F.R.S.

Rather more than thirty years ago I gave a series of demonstrations at the Royal College of Surgeons of England on specimens—some 250 in number—illustrating congenital malformations of the face.[1] Amongst them was a form, depicted in *Figs.* 84, 85, which I found difficult of explanation. A furrow or groove is seen to pass from the angle of the mouth; it may reach the external auditory meatus as in *Fig.* 84, or it may cease at or near the anterior border of the masseter as in *Fig.* 85. In *Fig.* 84 the groove is still open, and has a pink lining membrane.

Fig. 84.—Photograph of a child of 14 months showing a congenital groove on right cheek. (*G. H. Edington's case.*)

Fig. 85.—Photograph of a boy in which the groove shown in *Fig.* 84 has become filled by scar tissue. There is a tag or appendage growing from the scar. The tragus is divided (supernumerary auricle). (The letter which accompanied this photograph has been mislaid, so that I am unable to acknowledge its donor.)

In *Fig.* 85 the groove has been filled up by cicatricial tissue; a tag-like appendix grows from the scar; the tragus of the ear has an accessory cartilaginous process placed beside it. As is usual in such cases there is in *Fig.* 84 an open fissure at the outer angle of the mouth; the groove continued the line of the fissure.

These small pedunculated processes of skin are often present. *Fig.* 86, taken from a photograph loaned to me by the late Prof. William Wright, shows a child with such a process (1) and the position of processes in two other cases (2, 3). In Dr. Wright's case, too, the tragus shows a division or accessory auricle (4). In *Fig.* 87 these tags are represented by an 'amniotic adhesion'—which after encircling the left arm ends on the inner surface of the placenta, where it is joined

by another 'adhesion' which issues from an occipital meningocele. This case was reported by von Winckel.[2]

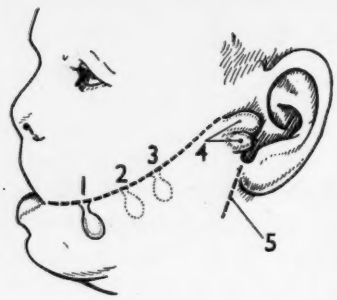

FIG. 86.—Taken from the photograph of a child in which there was a congenital crease crossing the left cheek from mouth to ear. 1, A cutaneous tag; 2, 3, The position of tags on two other cases; 4, A divided tragus; 5, The position of the ear (first) cleft—were it to persist.

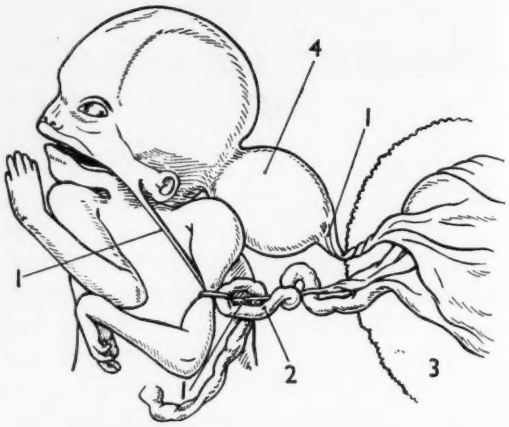

FIG. 87.—A fibrous band (amniotic adhesions arising along the site of an ear-mouth congenital groove in a 5½ months fœtus. Another band is of similar nature and arises from the apex of an occipital meningocele. 1, Band; 2, Umbilical cord; 3, Placenta; 4, Meningocele. (*After von Winckel.*)

STREETER'S FŒTAL DYSPLASIA

Many other cases have been reported[3], but so far no satisfactory explanation of their production has been given. The explanation I am now to offer is based on a research carried out by Dr. George L. Streeter,[4] who has presided over the Department of Embryology of the Carnegie Institution, Washington, for many years and whose important contributions to our knowledge of the development of the human body deserve to be better known to surgeons than they now are. By 1930 Dr. Streeter had at his disposal 16 cases of a condition he speaks of as "focal deficiencies in fœtal tissues", but which, as its aetiology is as yet imperfectly understood, I propose to name, in the meantime, 'Streeter's fœtal dysplasia'. It is the source of many forms of congenital malformation. Dr. Streeter is inclined to ascribe the lesions which become manifest in the fœtus as it develops to a fault inherent in the chromosomes of the fertilized ovum. This may be so, but in the meantime, for reasons I shall adduce later, it is more profitable for us to regard the fault as lying in a temporary—or permanent—breakdown in the circulatory system of the fœtus.

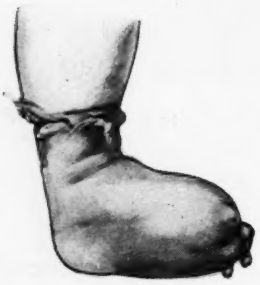

FIG. 88.—The medial aspect of the left foot and leg of the fœtus described in the text. (*After Streeter.*)

Let us examine the outward signs of Streeter's dysplasia as manifested in his youngest case, a male fœtus, 14 weeks of age (sitting height 86 mm.), removed by operation, with membranes intact. The mother had suffered from uterine fibroids and metrorrhagia; the fœtus had been dead some weeks before the date of operation; the placenta was "delicate and easily torn". In *Fig.* 88 is shown the left foot and leg of this fœtus. The distal parts of all the toes have been lost by a

process of 'fœtal necrosis'; round the leg, just above the ankle, is a circular necrotic groove filled with exudative tissue in which there is embedded a constricting ring or anklet of fibrous tissue. On the right leg there is a corresponding groove and ring, but the toes are intact. The anklets of the two sides had become joined, so that the legs were bound together. Three fingers of the left hand and all the digits of the right hand were encircled by fibrous wrappings of the same exudative material and the parts distal to the wrappings were undergoing separation and necrosis. Clearly we are witnessing a stage in the process which gives rise to intra-uterine amputations.

In *Fig.* 89 a section along the right leg of this fœtus is given to show the arrangement of parts in the groove which contains the constricting ring of fibrous tissue. On one side (A) the groove is occupied by a loosely organized exudate,

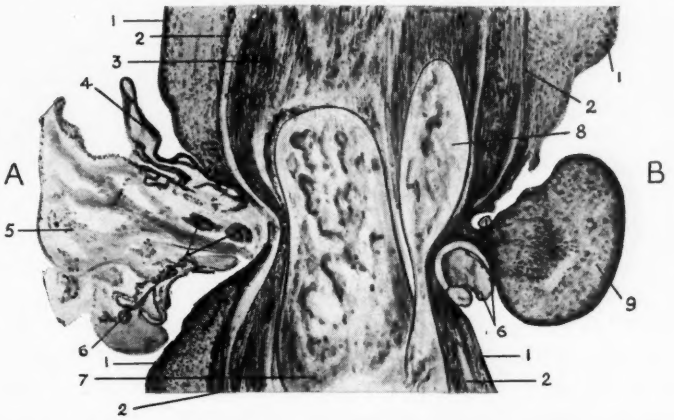

FIG. 89.—Section along the right leg of the same fœtus to show the necrotic annular groove and the structures contained within it—a diagrammatic rendering of Dr. Streeter's illustration (Fig. 14, Plate I). 1, Cutis; 2, Deep fascia; 3, Muscle; 4, Epidermis; 5, Organized exudate; 6, Bands; 7, Tibia; 8, Fibula; 9, Tag. For further explanation, *see* text.

near the base of which two strands of the constricting fibrous ring are seen on section. The base of the mass springs from a necrotic layer of fascia—under which there is a bending in and compression of the muscular stratum. The epidermis has become detached—but above A a remnant is seen to descend to the bottom of the groove—which had been covered with cutaneous epithelium. When the necrotic layer of fascia, with which the exudative mass in the groove is continuous, is traced upwards and downwards it is seen to become continuous with both the cutis and the deep fascia, these two being widely separated, both above and below the groove, by subcutaneous tissue. At the bottom of the groove, then, cutis and deep fascia become fused into a single fibrous stratum; on the other side of the leg (B) cutis, deep fascia, muscle, and periosteum are fused into one stratum. The stratum on this side of the leg is not necrotic but remains very much alive and has grown out to form a tag-like appendage, covered with epithelium, and in every way comparable to the dermal tags found springing from the buccal grooves of the face. At each side of the base of the tag and within the groove are seen sections of the annular constricting fibrous band.

If we examine the lesion in the hands of this fœtus we shall obtain more light on the peculiarities of fœtal dysplasia. The initial lesions lead on to intra-uterine

necrosis and amputation. In *Fig.* 90 a dorsal view of the left hand is depicted; three digits are wrapped in fibrous exudate; at the proximal margin of the fibrous bands a line of separation has been demarcated. *Fig.* 91 is taken from Dr. Streeter's section of the right hand, which exposes a longitudinal view of the thumb. All

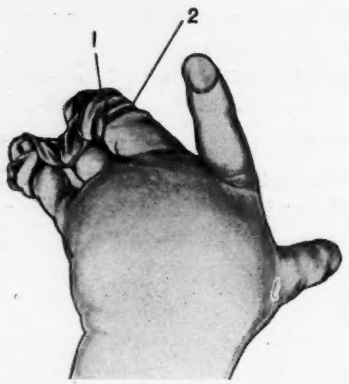

FIG. 90.—Dorsal aspect of the left hand of the fœtus described in the text. 1, Fibrous band; 2, Line of separation. (*After Streeter.*)

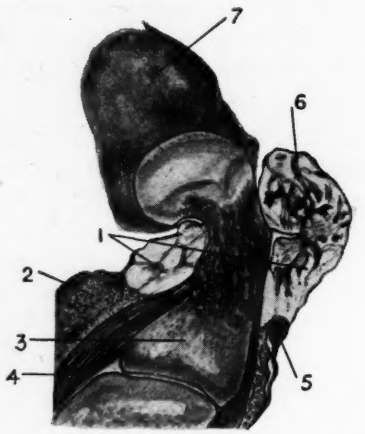

FIG. 91.—A longitudinal section of the thumb of the right hand of the same fœtus, showing the tissues ending in a fibrous ring which surrounds the proximal phalanx. The distal part of the thumb is semi-necrotic. On the dorsal aspect of the thumb the fibrous ring is made up of two parts: the distal part surrounded an adjacent digit. 1, Band; 2, Subcutaneous tissue; 3, Proximal phalanx; 4, Tendon; 5, Epidermis, dorsal aspect; 6, Band of 3rd finger; 7, Distal part of thumb. (*Diagrammatized from Dr Streeter's illustration,* Fig. 15, Plate I.)

FIG. 92.—Left foot of a female fœtus, 27 weeks of age, illustrating a stage in the progress of Streeter's dysplasia. 1, Scar; 2, Band; 3, Crease. (*After Streeter.*)

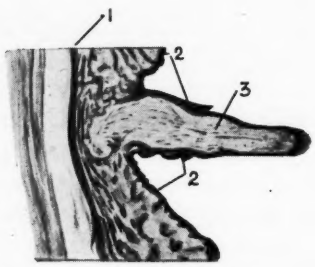

FIG. 93.—Section across crease shown in *Fig.* 92, made so as to expose the fibrous (amniotic) band, also shown in *Fig.* 92. 1, Deep fascia; 2, Epidermis; 3, Band.

the soft tissues of the thumb are seen to end opposite the middle of the proximal phalanx in a circular mass of organized fibrous tissue; the part of the thumb distal to the fibrous ring is disorganized and on its way to being shed. The epidermis is seen, on the dorsal aspect of the thumb, to end in a thickening at the proximal base of the fibrous ring. The fibrous ring on the dorsal aspect of the thumb is adjacent to, and partly adherent with, a constricting band which surrounded an adjacent finger.

A later stage in the progress of Streeter's dysplasia is illustrated by the specimens shown in *Figs.* 92, 93. They are taken from a case of Dr. Streeter's—a fœtus of

about 27 weeks, and therefore some 13 weeks older than the fœtus we have just been describing. In the present instance the right foot had become shed or amputated above the ankle and the stump was healing. The amputated foot was found in the cavity of the amnion; in size it corresponded to that of a fœtus of about 16 weeks. The left foot of this case is shown in *Fig.* 92. Above the ankle there is a transverse crease from which issues a fibrous cord. A section along the base of this cord and across the crease is shown in *Fig.* 93. The base of the cord appears to issue from the deep fascia and passes through the epidermis, finally becoming devoid of epithelial covering. The more superficial fibres of the cord are nucleated. It is also important to note other pathological lesions in the left foot—namely, scarred tissue on the medial side of the ankle and in the subcutaneous tissue over the dorsum of the foot, which is dense and contracted. Digits in both hands are embedded in fibrous bands; the distal parts of some of the digits have been shed.

Such then are the external manifestations of Streeter's dysplasia. The condition at the end of the 6th month of fœtal life shows many changes from those we have noted at the end of the 3rd month. But even at the end of the 3rd month the dysplastic condition must have reached an advanced stage. Circumstantial evidence points to the end of the 2nd month of development as the period of incidence. It is then that toes and fingers, the most common sites of this disorder, are just assuming their discrete shapes.

We have noted Dr. Streeter's opinion that the defects just described are due to a developmental factor inherent in localized areas of tissue in the distal parts of the limbs of afflicted fœtuses. Were a surgeon to approach such cases and note the signs with which he is familiar in cases of gangrene, he would immediately suspect a breakdown in the supply of blood to the developing extremities. He would the more readily look for a temporary or permanent failure in the general circulatory system seeing that all four limbs are usually affected to a greater or less extent. He would examine the placenta and umbilical cord for evidence of such a failure and if he did so in Dr. Streeter's cases he would find in nearly every one evidence of defect and circulatory failure. At the end of the 2nd month of development, which appears to be the period at which fœtal dysplasia of the limbs usually begins, the placenta is undergoing a rapid expansion and is therefore in its most vulnerable phase of growth.

Gangrene of the hands and feet of a fœtus of the 3rd month follows a totally different course from that seen in the limbs of an adult. In the first place, the tissues of the fœtus are less dependent on blood-supply for health and growth than are the same tissues in an adult. The human heart begins to beat about the 24th day; until then the tissues are dependent on lymph seepage; it is not until towards the end of the 2nd month that a cardiac circulation is fully established. Fœtal tissues have a higher degree of vitality and individual response than have mature tissues. Some fœtal tissues, such as the fibroblasts of the skin and deep fascia, respond in a particularly vigorous manner when cut off from their blood-supply. In *Figs.* 89, 91, 93, the fibrous outgrowths, which are formed at lines of partial or of complete separation of tissues, spring from the deep fascia.

In the second place, fœtal limbs are suspended in amniotic fluid, and kept at body temperature, circumstances which are, as Dr. Streeter has pointed out, particularly favourable for 'tissue culture'. When the rings of separation first appear on digits and distal parts of the limbs, there must be a free exudate of

lymph along the lines or grooves of tissue separation. Into these grooves of separation fibroblasts of the deep fascia and cutis spread and there multiply, thus forming fibrous cords or bands. Even along such a fibrous process as is depicted in *Fig.* 93 there must be a seepage of lymph from the deeper tissues of the fœtal limb. In this way are formed all of these pathological formations known as 'amniotic bands'; all of them are formed from and by the fœtus at sites of tissue injury.

We have made this long detour in our narrative in order to clear up the nature of certain anomalous fissures, creases, and integumentary processes which occasionally mar the human face. Before returning to a consideration of these deformities, however, there are still two other embryological researches we must take into consideration.

VASCULARIZATION OF THE EXTERNAL COVERINGS OF THE HEAD

One of these researches is by Miss Ellen B. Finley,[5] a pupil of Prof. Florence Sabin. In *Fig.* 94 I take the liberty of reproducing one of her illustrations, because

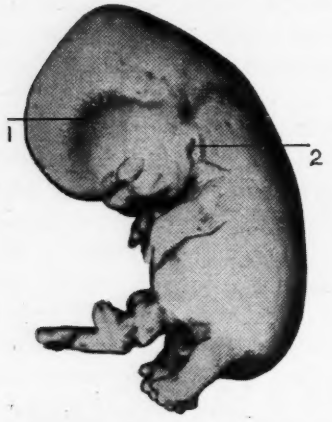

FIG. 94.—Profile of a fœtus 23 mm. in length and in the 8th week of development. The process of vessel formation (angiogenesis) which leads to vascularization of the scalp is seen spreading upwards in the temporal and occipital regions of the head. 1, Vascular wave; 2, Ear. (*After Ellen B. Finley, 'Contributions to Embryology'*, 1922.)

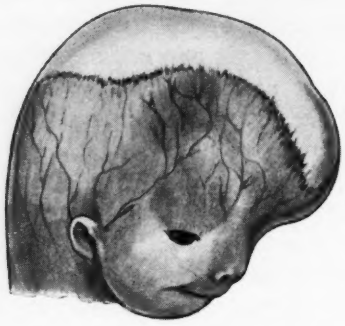

FIG. 95.—The point reached by the wave of scalp vascularization in the 9th week. (*After Miss Finley.*)

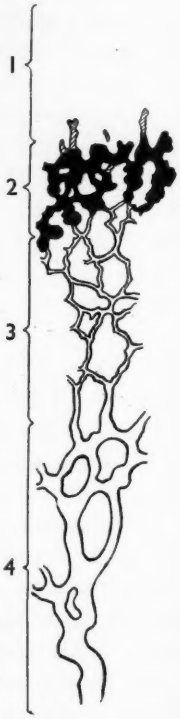

FIG. 96.—To illustrate the manner in which the scalp becomes vascularized. 1, Avascular zone; 2, Angioblastic zone; 3, Capillary plexus; 4, Definitive vessels. (*After Miss Finley.*)

it depicts a human fœtus at the stage of development which marks, as we suspect, the onset of Streeter's focal dysplasia. The fingers are already formed; the toes are in process of demarcation. Two waves of vascular tissue, one in front of the ear, the other behind, are ascending on the temporal and occipital regions of the head of the fœtus; above these waves the scalp and coverings of the skull are still devoid of a blood-supply. Hochstetter of Vienna drew attention to this vascular invasion of the scalp in 1916; Miss Finley investigated the mode and rate of its growth. I can best summarize her observations by drawing the reader's attention to *Figs.* 95, 96, which are based on her original illustrations. *Fig.* 95 shows the line reached in the scalp of a fœtus a week older than that depicted in *Fig.* 94. A week later still—that is, about the end of the 10th week—the waves

of invasion on each side of the head will have met along the midline, thus completing the vascularization of the scalp. It is important to note the manner in which the growing margin of the wave is advanced. Miss Finley found that the mesenchyme of the scalp, just in advance of the growing margin, becomes transformed into red blood-cells and vascular endothelium; the scalp is not invaded by new vessels sprouting from the old, but provides the material for vascular extension.

Let us suppose, for a moment, that during the extension of the vascular network into the scalp, a partial and temporary breakdown of the placental circulation should take place; then we may justly infer that it would be the growing margin of vascularization that would suffer the chief damage. Prof. H. A. Harris[6] has demonstrated that the growing ends of long bones reflect the illnesses of childhood, for instead of normal bone being laid at the growing disc a dense plate is formed. At the growing margin of vascularization of the scalp dense fibrous tissue results, giving rise to congenital defects of the scalp (*Fig.* 97). We may transfer these observations to the dysplasia described by Dr. Streeter in developing feet and hands, for, as we have seen, the initial damage falls on subcutaneous strata, corresponding to those of the scalp. At the end of the 2nd month of development the distal parts of the human extremities—hands and feet—are still in the capillary stage of vascularization.

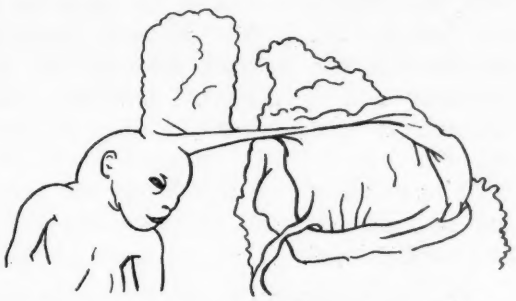

FIG. 97.—The most common type of amniotic adhesion—passing from a meningeal mass to the amnion. For the origin of such bands, *see* p. 181. (*After Streeter.*)

CONGENITAL DEFECTS IN THE MID-DORSAL LINE

The other embryological research I have to touch upon is that made by my old friend Dr. N. W. Ingalls.[7] He has described a series of human fœtuses, which were in the 2nd and 3rd months of development, all of them showing plastic defects along the mid-dorsal line of the body. So far as the clinical histories of these fœtuses are available they point to a circulatory failure, with cystic villi in the placenta, a more or less avascular condition of the umbilical cord, hydramnios, then death in the 2nd or 3rd month of development, with expulsion from the uterus some weeks after fœtal death. Along the midline of the fœtal head Dr. Ingalls noted blebs or collections of fluid which may be situated under the epidermis or more deeply, in the subdermal tissues. He noted in particular that areas of the fascial tissues along the mid-dorsal line of back and head were behaving in a way very similar to that of fascial tissues at the lines of separation in Streeter's dysplasia. Dr. Ingalls also called attention to the fact that the dysplasia may not take the linear form seen in the digits and ankle, but may involve an irregular area of dermal and subdermal tissues of greater or less extent. Dr. Streeter noted such areas on the limbs of several of his cases (*see Fig.* 107).

The activities and responses noted in dermal and subdermal tissues along the midline of the head and back of the fœtuses described by Dr. Ingalls are of

the same nature as those we have noted in Streeter's dysplasia. As further evidence in support of this inference I call attention to the occurrence of a circulatory (placental) failure, the occasional occurrence of amniotic adhesions, and the fact that dysplastic lesions of the digits and feet may accompany the midline changes described by Dr. Ingalls. We infer that the failure of circulation in such fœtuses as have just been noted will fall first and most severely on the vascularization of the subdermal tissues of the midline which takes place during the later weeks of the 2nd month and the earlier part of the 3rd month.

Meningoceles.—Meningoceles are structures of obscure origin; they occur along the mid-dorsal line of the head, the two most common sites being occipital and frontal. Dr. Ingalls' research seems to me to throw light on the origin of meningoceles and to bring them into the same category of lesions as those which we have seen in Streeter's dysplasia. In *Fig.* 98 we have copied one of his illustrations—a side view of a fœtus at the end of the 2nd month of development and showing a 'bleb' situated in the midline of the occipital region. The covering of the bleb is translucent and sub-ectodermal in origin; blood has leaked into its fluid content. The chorionic villi were found to be "swollen, irregular, and smaller cystic forms are quite plentiful", from which we infer there must

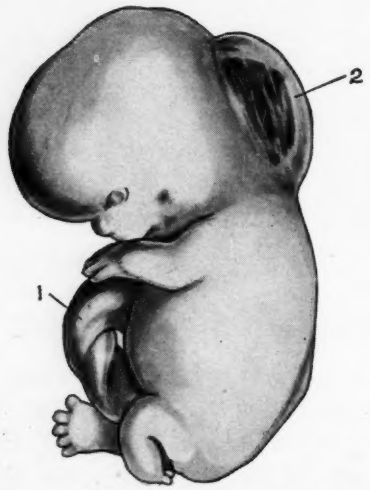

FIG. 98.—Profile view of the left side of a fœtus at the end of the 2nd month of development (25 mm. in length) in which there is a large subepidermal bleb over the region of the 4th ventricle. 1, Umbilical cord; 2, Bleb. (*After N. W. Ingalls.*)

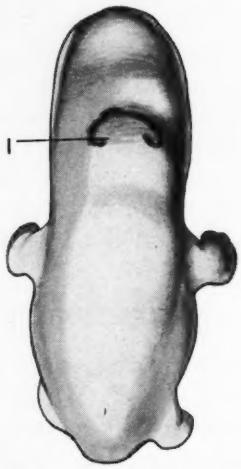

FIG. 99.—Dorsal view of a pathological embryo, 18·5 mm. in length, but showing evidence of arrested development, in which there is a dysplastic area over the 4th ventricle. 1, Dysplastic area. (*After N. W. Ingalls.*)

have been a defective placental circulation. Suppose the circulatory breakdown proved to be temporary and that the fœtus continued to develop, what would have happened to the occipital bleb? I take it that its walls would have collapsed and that the dermal and subdermal tissues thus exposed would have formed a weak fibrous wall over the 4th ventricle—the possible site of a meningocele.

The fœtus depicted in *Fig.* 99 presents us with a different and earlier lesion. This fœtus shows many signs of arrested development and represents a stage which is younger than that seen in *Fig.* 98 by about two weeks. "In the dorsal midline", to quote Dr. Ingalls's words, "about the region of the lower rhombencephalon, there is a small, transversely elongated, somewhat smoother, discoloured

area, slightly greenish in colour. It is roughly reniform in outline, sharply marked from the surrounding parts, slightly elevated and measures about 3 by 1 mm." The discoloured area, when sectioned, showed extensive histological changes, especially in the connective tissue immediately under the discoloured epithelium. The densely cellular fibroblastic area was sharply circumscribed. The fibroblastic reaction seen here seems to be of the same nature as that which Dr. Streeter has described in connexion with necrosis of the digits. If hydrocephalus were to set in, as it often does in fœtuses which are the subjects of the lesions we are dealing with, then this area over the roof of the 4th ventricle would provide a favourable site for the origin of an occipital meningocele. We can understand, too, why an exudate occurs in lesions which are accompanied by the formation of "amniotic adhesions" of the kind illustrated in *Figs.* 87 and 97. In the case of the fœtus depicted in *Fig.* 99 there must have been a circulatory collapse, for the chorion was "very thin, villi were absent save in one area" and "no vessels were to be seen anywhere".

CONGENITAL FACIAL DEFECTS

Buccal Grooves and Creases.—We are now in a position to return to a consideration of the congenital grooves and scars which cross the human cheek—from the angle of the mouth towards the ear—which they may reach. When they do reach the ear the tragus is usually subdivided (*see Fig.* 86). The appearance and character of these grooves are similar to those presented by the feet and hands in cases of Streeter's dysplasia, and to the lesions described by Dr. Ingalls in the

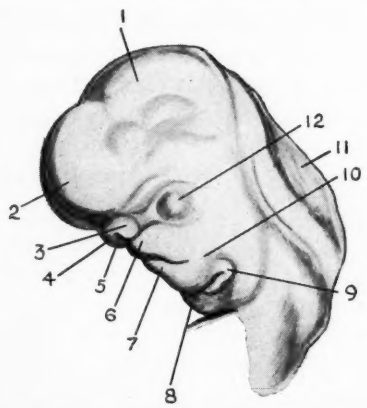

FIG. 100.—The point reached in the development of the face of the human embryo towards the end of the 6th week (10 mm. in length). As seen in profile. 1, Mid-brain; 2, Cerebral vesicle; 3, Lateral nasal process; 4, Mesial nasal process; 5, Nose; 6, Maxillary process; 7, Mandibular process; 8, Hyoid process; 9, Ear; 10, Cheek; 11, 4th ventricle; 12, Eye. (*After Professor J. E. Frazer.*)

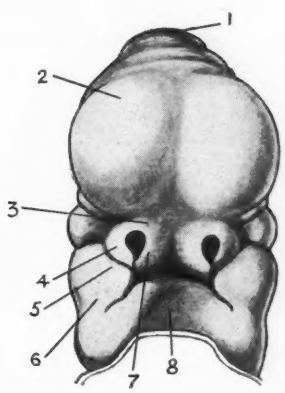

FIG. 101.—The same embryo seen in full face. 1, Mid-brain; 2, Cerebral vesicle; 3, Mesial nasal process; 4, Lateral nasal process; 5, Maxillary process; 6, Cheek; 7, Fronto-nasal process; 8, Mandibular process. (*After Professor J. E. Frazer.*)

scalp and along the mid-dorsal line of the head and spine. The integumentary tags which spring from facial grooves are of the same nature as those which may be found in grooves of separation at the ankle (*see Fig.* 89). Further, it will be clear from *Figs.* 100, 101 (which represent the condition of the mandibular and maxillary processes in the face of a human embryo towards the end of the 6th week of development) that these two processes spring from a common base which occupies

all the space between the eye and ear, this common area representing the groundwork of the cheek. It is true that the angle of the mouth makes an approach to the ear, but as development proceeds the mouth is advanced by growth of tissue within the fœtal cheek, not by a union of the adjacent free borders of the maxillary and mandibular processes. In certain cases the whole thickness of the cheek may necrose, leaving part or the whole an open cleft from mouth to ear. An open condition of the cleft is spoken of as *macrostoma*.

The buccal grooves or creases, then, do not represent the edges of an ununited embryological cleft, such as occurs in hare-lip, but have to be accounted for in some other manner. As we shall see later, Dr. E. Wolff[8], on subjecting the heads of fowl embryos to measured exposures of X rays, thus damaging the tissues which go to form the maxillary and mandibular processes, produced a superficial defect similar to the congenital lesion we find on the human cheek. In the chick the chief defect lies in the superficial or dermal structures—just as is the case in mandibular maxillary creases of the human fœtus. The congenital crease which appears on the human cheek lies between two developmental territories—between the mesoderm of the mandibular and of the maxillary processes. Each fœtal process has its own independent blood-supply; that of the maxillary process being the internal maxillary artery, while the mandibular is supplied by the vessel which becomes subsequently the inferior dental. The congenital linear lesions of the cheek appear to develop along the line at which the vascularization of the mandibular process meets with that of the maxillary process. It is along lines of vascular union that corresponding defects are apt to occur in the dorsal midline of the body where right and left processes of vascularization meet. When we find the buccal crease at its earliest point of development I expect it will show a superficial or complete linear necrosis, with exudate, the formation of fibrous bands, amniotic adhesions, healing, and integumentary tags (*see Fig.* 87). In brief, I regard buccal creases as a facial manifestation of Streeter's dysplasia. The facial artery which crosses the ventral ends of the mandibular and maxillary territories in the later months of fœtal life, is a secondary formation.

FIG. 102.—A median cleft of the lower lip and mandible of a child aged 4 months, illustrating a dysplastic lesion at the junction of the ventral ends of the right and left mandibular arches. (*Teratological Collection, R.C.S. Museum, No.* 244.05.)

Median Cleft of Lower Lip and Mandible.—In *Fig.* 102 is depicted a case of median cleft of the lower lip and of the mandible, which occurred in the practice of Sir Wm. MacCormick, of Sydney; he presented a model of the case to the Museum of the Royal College of Surgeons of England. As will be seen in *Fig.* 101, the ventral ends of the mandibular arches are not normally separated by a cleft; being formed within the lower margin of the embryonic mouth (stomodæum). This is a midline lesion of the same kind as Dr. Ingalls described in connexion with the midline of the dorsum of the head and back. The margins of the cleft have the cicatricial appearance of dysplastic lesions. Schwalbe[11] figures a similar case and also one in which the fissure has undergone cicatricial healing.

Oro-temporal Cleft.—In *Fig.* 103 an uncommon form of dysplastic lesion is illustrated. It crosses the right cheek of a full-time fœtus, passing from the angle of the mouth to end in a wide depression in the right temporal region, where the floor is formed by a dense membrane, representing all the tissues on the lateral wall of the head from skin to dura mater; a similar cleft crosses the left cheek. It will be remembered that in severe cases of Streeter's dysplasia of the extremities all the tissues, from skin to bone, are involved (*Fig.* 89). So it is here. It will be observed that this fissure crosses the territory of the maxillary processes; the lesion does not fall, as others we have described, at the junction of two territorial areas. A similar fissure is illustrated in *Fig.* 112.

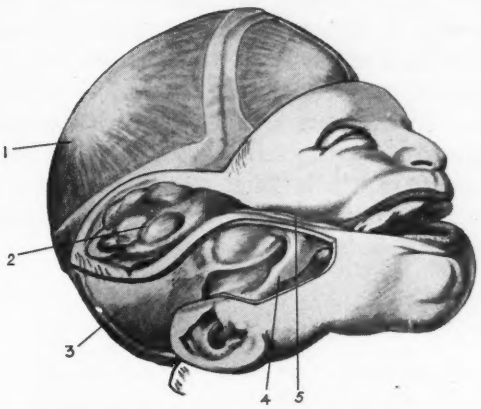

FIG. 103.—A dysplastic lesion crossing the cheek of a full-time child from the mouth to the right temporal region. 1, Upper parietal area; 2, Temporal area of cleft; 3, Lower parietal area; 4, Zygoma; 5, Buccal part of cleft. (*Teratological Collection, R.C.S. Museum, No.* 202.92.)

Extreme Foetal Defects.—We now pass on to the consideration of dysplastic lesions of the face which are accompanied by maldevelopments of the facial elements to such extreme degrees that they cease to concern us from a surgical point of view, and are mainly of pathological interest. In *Fig.* 104, which has been drawn from a full-time fœtus (No. 356 in the Teratological Collection of the Museum of the Royal College of Surgeons of England), the various processes which grow into the stomodæum to form the face have been arrested at an early stage of development,

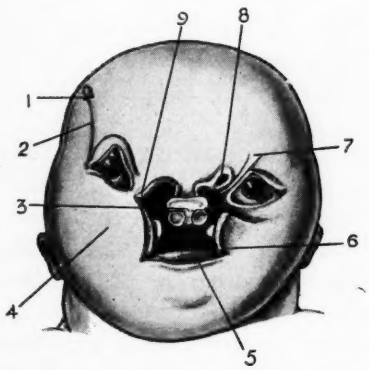

FIG. 104.—Dysplastic and other malformations in the face of a full-time fœtus. 1, Integumentary tag, at the end of a dysplastic crease (2), which begins at the outer canthus of the eye; 3, Fronto-nasal process carrying median part of the upper lip and premaxilla; 4, Right maxillary process; 5, Lower lip; 6, Left maxillary process; 7, Fibrous, dysplastic band; 8, Left nostril and nasal process; 9, Right lateral nasal process.

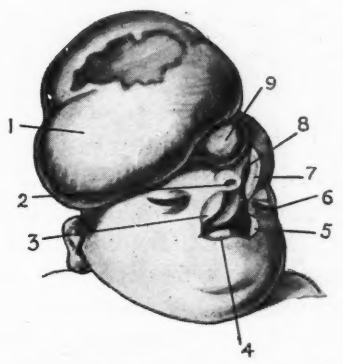

FIG. 105.—Face of full-time fœtus with ectopic cranial membranes and a dysplastic malformation of the face. 1 and 9, Massed ectopic and dysplastic membranes of brain; 2, Right nostril; 3, Right maxillary process; 4, Lower lip; 5, Left maxillary process; 6, Left eye; 7, Left lateral nasal process; 8, Dysplastic furrow between the left lateral and left mesial nasal processes, ending above in the ectopic mass of membranes.

a wide cleft, separating the maxillary and nasal processes, remaining below and to the mesial side of the left eye. Extending from the outer canthus of the right eye there is a trace of a crease which ends in a tag situated in the right temporal region.

This dysplastic crease lies at the junction of two vascular territories, namely, the frontal, supplied from the ophthalmic branch of the internal carotid, and the superficial temporal, from the external carotid. A fibrous band is seen to cross the left upper eyelid—an additional sign of dysplasia. This specimen also interests us from another point of view. The dysplastic lesions are accompanied by a severe arrest in the process of facial development. Complete failures in the union of the nasal and maxillary processes are so frequently combined with dysplastic lesions (*Fig.* 105) that we may suspect that they share in the same pathological disturbance.

In evidence of the statement just made, I cite two more of Dr. Streeter's cases. One of these is illustrated in *Fig.* 106; it is that of an infant, 5 months of age, in which there is double hare-lip and cleft palate, with dysplastic lesions in fingers and toes. These lesions I have supposed to make their appearance towards

FIG. 106.—A, Face of an infant of 5 months, showing non-union of nasal and maxillary processes: 1, Right lateral nasal process; 2, Skin of median part of upper lip, and 3, the premaxilla—both formed in the fronto-nasal process; 4, Maxillary process. B, Fingers of the right hand showing constrictions on fourth and fifth digits. C, Digits of the right foot; the distal part of the great toe has undergone intra-uterine amputation, while the distal parts of the second and third toes have been partially destroyed.

the end of the second month of development, but if the facial clefts and the constriction of the digits arise from the same pathological state (a breakdown in the placental circulation) and at the same time, then the onset is earlier than I have stated. For if reference is made to *Fig.* 101, which shows the point reached in the development of the face at the end of the 6th week, then we have to go back to this early stage to account for the facial cleft.

In *Fig.* 107 there are depicted associated lesions in another of Dr. Streeter's fœtuses. The case is that of a full-time, stillborn fœtus with massed ectopic membranes on the head and a nasal cleft or furrow—almost a replica of the condition shown in the specimen depicted in *Fig.* 105, save that the nasal furrow lies along the right side of the nose instead of on the left side, as in the previous case. With these lesions in the head of the case described by Dr. Streeter, there were associated typical dysplastic lesions of both feet and of the left hand. The condition of the left foot and leg is represented in *Fig.* 107, B. There is a deep annular crease or furrow, which fuses at its inner limits with the deep fascia of the leg, but its sides are now lined with normal skin. The great toe, which had undergone

intra-uterine amputation in the right foot, is practically intact, but the distal parts of the second and third toes are in process of separation or have separated. There are also cicatricial areas on the dorsum of the foot and on the extensor aspect of the leg, indicated in *Fig.* 107, B.

Anencephaly.—Now the dysplastic lesions of the extremities are associated with ectopia of the brain with a sufficient frequency to raise the suspicion that they may be manifestations of the same underlying pathological disturbance occurring during the development of the fœtus. If this is so, and if all the lesions are simultaneous in onset—which they need not be—then we have to seek for the disturbance at a still earlier stage of development. The arrest which brings about anencephaly or ectopia of the brain must be traced back to defects occurring in the 4th week of development.

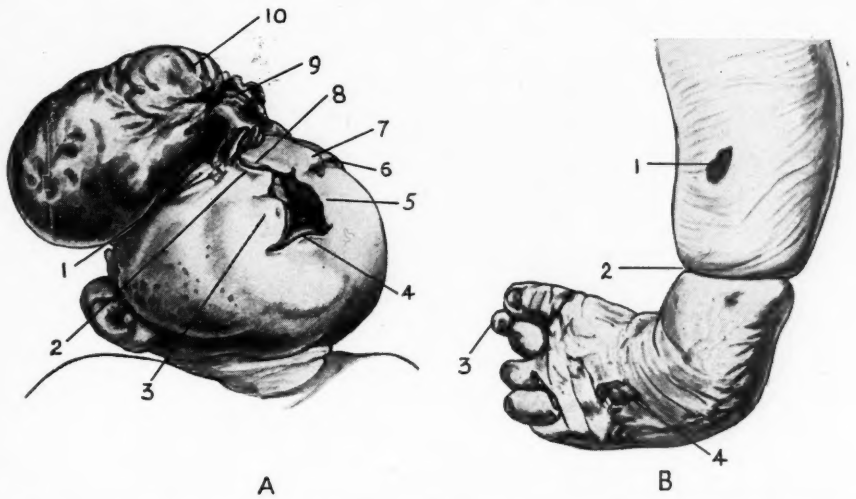

FIG. 107.—A, Head and face of a full-time fœtus, showing the membranes of the brain massed and exposed on the head, with a dysplastic furrow or depression ascending in the nose from the mouth to the exposed membranous mass: 1, Right eye; 2, Right lateral part of nose; 3, Right maxillary process; 4, Lower lip; 5, Left maxillary process; 6, Left eye; 7, Median and left part of the nose; 8, Dysplastic furrow; 9, 10, Ectopic membranous masses. B, Left leg, showing: 1, Dysplastic area on leg; 2, Annular dysplastic furrow; 3, Dysplastic lesions of toes; 4, Dysplastic area on dorsum of foot.

Dodds and De Angelis[9] have described a human fœtus showing a very early stage in the development of anencephaly; their description helps us to understand some of the lesions we are dealing with. A profile view of their specimen is reproduced in *Fig.* 108, and a dorsal view in *Fig.* 109. The fœtus is only 16·5 mm. in length and is probably at the end of its 2nd month, yet development of the brain has been arrested at the stage reached in the 4th week, when the neural plates, which should become enclosed to form the brain, and the mesodermal tissue, which should provide membranous and cranial coverings, lie exposed on the cephalic extremity of the embryo.

We have here a repetition of the same kind of failure which at the caudal end of the embryo gives rise to spina bifida. Our authors found, as occurs in cases of spina bifida, that all the issuing cranial nerves and their attached ganglia had been formed with the exception, in this case, of the 8th nerve. In the face they found a double hare-lip and the limb buds showed some degree of arrest. Almost the

whole of the neural plate, which here remains exposed to the action of the amniotic fluid, has disappeared, only one area remaining attached to the underlying mass which represents the membranous coverings of the brain. In our illustration an interrupted line (4) marks the junction of skin with the membranous vascular masses exposed on the head of this fœtus. In the latter part of the third week and earlier part of the fourth, had development pursued its usual course, these lines, now situated on the sides of the head, would have met along the mid-dorsal line and embryological union or ' healing ' would have taken place.[10] When a section is made across the line at which the skin meets with the membranous mass—at a later stage than that represented in *Figs.* 108, 109—the appearance presented is very similar to that

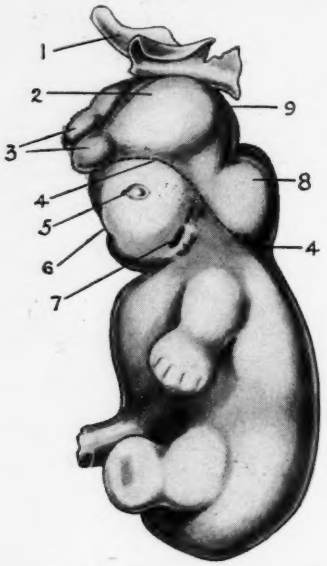

FIG. 108.—Profile of a human fœtus, 16·5 mm. in length and at about the end of the 2nd month of development. 1, Detached part of the neural plate—the only part that was found; 2, Masses representing membranes of the thalamencephalon; 3, Masses representing the cerebral membranes; 4, Line along which skin meets with capsular tissues of skull and brain; 5, Eye; 6, Median fissure of face; 7, Ear; 8, Membranous mass of hind-brain; 9. Membranous mass of mid-brain; 10. Funnel-shaped opening leading to central canal of spinal cord. (*Figs.* 108, 109 *after Dodds and De Angelis.*)

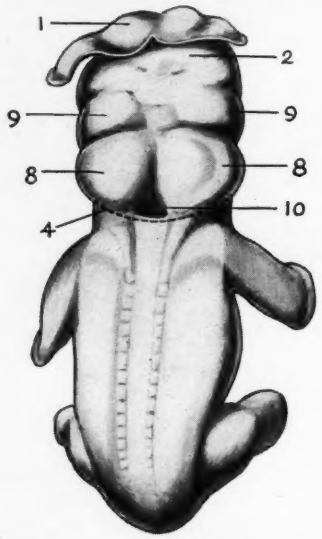

FIG. 109.—Dorsal view of the same fœtus. The numbers have the same significance as in *Fig.* 108.

met with in the dysplastic lesions described by Dr. Streeter (*Fig.* 110). At the bottom of the crease or furrow the subcutaneous tissue of the scalp is replaced by a dense fibrous stratum, which passes into the adjacent vascular membranous mass. Fibrous or desmoplastic bands are extruded from the mass. This marginal zone is the most common site of ' amniotic ' adhesions.

The dimensions of the vascular membranous masses which crown the heads of anencephalic fœtuses have often puzzled me. When we remember: (1) That the cerebral circulation is possible only within an enclosed capsule; and (2) That such vascular tissues exposed to the amniotic fluid at body temperature are thus placed in the most favourable circumstances for ' tissue culture '—then we can understand the changes which take place, particularly the formation of adhesive bands in the exudates of lymph. Anencephaly is so often combined with other forms of embryological failure, such as hare-lip, cleft palate, and other signs of Streeter's

dysplasia, that one may well suspect that the underlying pathology is the same for all—a nutritional disturbance. Now at the time the medullary plates are rising up

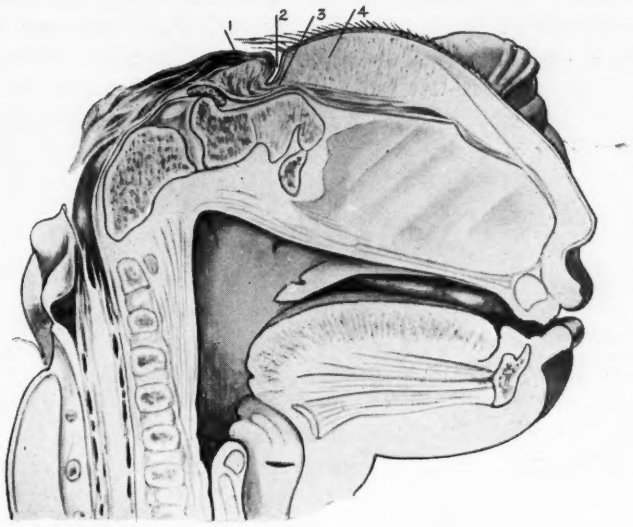

FIG. 110.—Section of the head of a full-time anencephalic fœtus at the junction of the skin and membranes. The section was made near the mid-line. 1, Membranes; 2, Groove; 3, Cicatricial tissue; 4, Scalp.

to be enclosed, the circulation of blood is only about to be initiated. In *Fig.* 111 I reproduce a case published by von Winckel[2] to illustrate the formation of 'amniotic adhesions' after the brain has become enclosed. It also throws light on the two cases depicted in *Figs.* 105, 107, where clefts of the nose are associated with anencephalic masses on the head. In *Fig.* 111 the fibrous bands pass up the forehead to end in tissue masses which are extruded on the crown of the head; each band commences at the margins of what was an olfactory pit and fold. Such bands I presume to be formed within necrotic grooves just as are the fibrous anklets and bracelets in Streeter's dysplasia. As the head expanded, these bands, being resistant, have dragged the olfactory pits towards the crown of the head.

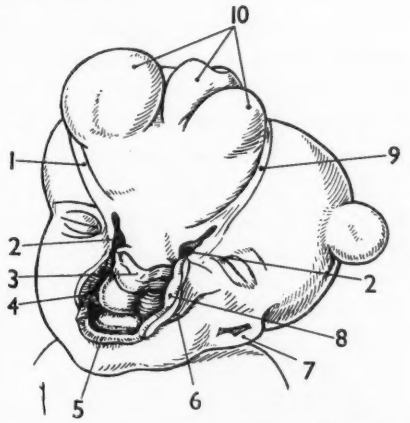

FIG. 111.—Head of human fœtus with symmetrical fibrous (amniotic) bands on the forehead, double hare-lip, and cleft palate. 1, 9, Right and left frontal bands; 2, 2, Right and left olfactory pits drawn up by bands; 3, Fronto-nasal process; 4, Right upper lip; 5, Lower lip; 6, Band formed in oro-orbital groove; 7, Left ear; 8, Left upper lip; 10, Extrusion masses (meningoceles ?). (*After von Winckel.*)

Why should necrotic grooves be formed along the line of these bands? The area between the bands is that supplied by the terminal branches (frontal) of the ophthalmic arteries. The areas outside the bands are supplied by the supra-orbital vessels and by the temporal branches of the external carotid. As in other cases of dysplasia of the face and head, the necrotic groove and bands are formed along marginal areas.

In *Fig.* 112 an anencephalic condition is combined with three examples of dysplastic fissure of the face. The case was recorded by Marchand.[11] On the right cheek a fissure passes from the angle of the mouth to the temporal region, as in the specimen depicted in *Fig.* 103; on the left cheek a fissure or crease ascends from mouth to orbit (as in *Fig.* 113), while a continuation into the left temporal

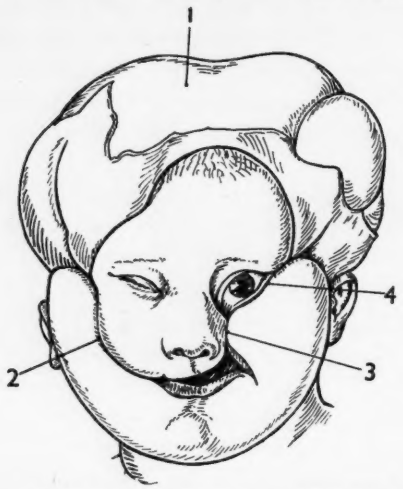

FIG. 112.—A case recorded by Marchand showing dysplastic fissures of the face. 1, Anencephalic masses; 2, Right oro-temporal fissure; 3, Left oro-orbital fissure; 4, Left fronto-temporal fissure.

FIG. 113.—Woman with dysplastic fissure (healed) passing from left upper lip to left lower eyelid. (*Schwalbe.*)

region corresponds to the temporal crease shown in *Fig.* 104. Here we have dysplastic fissures at three sites associated with an anencephalic condition.

Fissure from Lip to Eyelid.—There is no dysplastic lesion of the kind shown in *Fig.* 113 in the Museum of the Royal College of Surgeons of England; nor have I had the fortune to see one, and yet Schwalbe[11] depicts this condition in 5 cases. The scar line in *Fig.* 113 passes upwards on the right cheek from upper lip to lower eyelid, lateral to the position of the embryological groove in which the nasal duct is formed. Like the fissure shown in *Figs.* 103, 112, the scar line crosses the territory of that part of the cheek formed by the maxillary process, but in *Fig.* 113 it lies near to the junction of this process with the lateral nasal. Lateral to the scar is the area supplied by the infra-orbital branch of the internal maxillary artery, while medial to it is the nasal area fed by terminal branches of the ophthalmic artery. I suppose that the necrotic groove is formed along the junction of these two vascular areas.

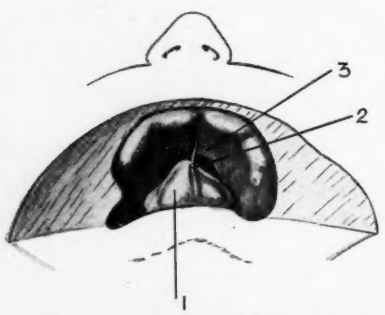

FIG. 114.—A fine fibrous band uniting the frænum of the tongue to partially cleft palate. 1, Tongue; 2, Band; 3. Palate. (*Teratological Collection, R.C.S. Museum, No.* 202.7.)

Band from Tongue to Palate.—*Fig.* 114 is a drawing made from a preparation in the Teratological Collection of the Royal College of Surgeons Museum. It represents the tongue and palate of an anencephalic fœtus in which the posterior part of the median fissure of the palate remained ununited: a fine fibrous band

passes from the palatal cleft to the frænum of the tongue. We have seen, in connexion with dysplastic lesions of the extremities, how neighbouring parts may become united by the organization of exudates of lymph. We have also seen that severe degrees of hare-lip and cleft palate may be associated with dysplastic lesions of the limbs and anencephalic conditions of the heads. This specimen supplies further evidence that a common pathology underlies all of these conditions. A case figured by Schwalbe[11] (Part III, p. 175, *Fig.* 114) shows a fibroplastic exudate in connexion with cleft palate.

DYSPLASTIC LESIONS IN MICE

Just before Dr. Streeter's monograph appeared Dr. Halsey J. Bagg[12] published an account of a breed of mice in which he had succeeded in producing dysplastic lesions of an hereditary nature. The mice, the subjects of his observation, were the progeny of normal mice which had been exposed to a series of brief doses of X rays, with the object of injuring their germ plasm. In the progeny of these mice appeared a proportion of descendants which suffered from the following lesions: small or absent eyes; kidneys absent on one or both sides, in some cases polycystic; dysplastic lesions of the feet. In about 80 per cent of cases of dysplasia of the feet there was no renal lesion; in 50 per cent there was no ocular lesions. It was not until he had inbred the dysplastic mice and had reached the eighth generation that Dr. Bagg began a systematic examination of the lesions of the limbs. He developed a technique which permitted him to expose the fœtus in the uterus, to mark its condition, to close the uterus, and so note the progress from, say, the 13th day, when lesions began to appear, until birth on the 20th day. For example, 67 embryos were exposed in the uterus of the mother on the 13th day—of these, 6 showed foot lesions in the earliest stage; 52 embryos were exposed on the 15th day—of these, 6 showed defects; on the 17th day, 18 of 75 had foot defects. Of 5200 embryos born during the period of observation (5 years) 432 suffered from dysplasia of the feet—rather more than 8 per cent. By mating parents showing extreme degrees of dysplasia he obtained a much higher percentage of defectives: in one case 3 out of a litter of 4.

Fig. 115 is a diagrammatized representation of an early stage in the development of a dysplastic lesion on the dorsum of the foot of a fœtal mouse. The section was made by Dr. Bagg on a fœtus which he removed from the mother on the 15th day of her pregnancy, and which corresponds to the stage of development reached by the human fœtus at the end of the 2nd month. A bleb has suddenly appeared in the subcutaneous tissue of the dorsum of the foot, the usual site, and contains a clear lymph, into which has just occurred an effusion of blood. The site of the effusion is seen at the base of the bleb (*Fig.* 115, 3) where fibroblasts appear to be in a state of activity.

This bleb on the dorsum of the mouse's foot is similar in all essential detail to that described by Dr. Ingalls in the occipital region of an early human fœtus and illustrated in this article by *Fig.* 98.

Dr. Bagg attributes the formation of such a bleb "to a localized arrest of development of part of the fœtal foot . . . associated with a perivascular lymph stasis." Now a localized arrest of development need not give rise to effusion of lymph and blood; only a localized necrosis of fœtal tissue could occasion such a

train of events, and the most probable cause of necrosis is a localized failure in the circulation of blood.

Nowhere has Dr. Bagg recorded the state of the placenta in these dysplastic embryos, although placental lesions, were they present, would not likely escape his vigilant eye. When the circulation of a fœtus falls to the lowest ebb, as in a parasitic twin, large lymph cysts or hygromata form in its subcutaneous tissues. This is best seen in a twin fœtus which has become dependent for blood-supply on the circulation of its host twin [13]. The active agent which brings about this localized anæmia and necrosis in *dysplasia fœtalis* still awaits discovery.

The stage reached a week later by the lesion shown in *Fig.* 115 is shown by another specimen illustrated in *Fig.* 116. The bleb is now replaced by a cap of dense necrotic tissue, made up of epidermis and hæmorrhagic débris. Around the margin of the necrotic area the cutaneous epithelium is dipping inwards; in the base of the area, where there is a small hæmorrhage, the fibrous tissue is actively proliferating towards the necrotic mass. Part of the distal end of the toe has become necrotic and been lost.

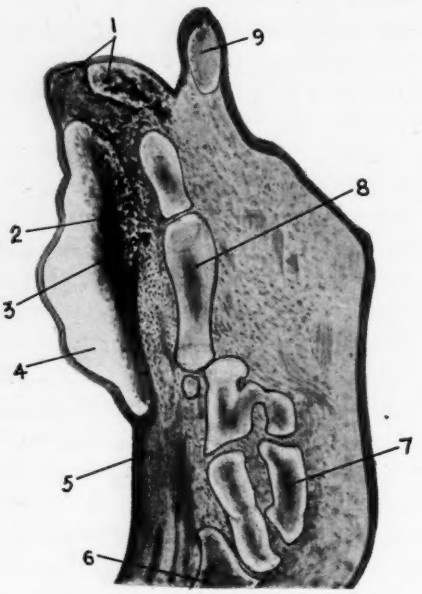

FIG. 115.—Longitudinal section of the right hind foot of a fœtal mouse, 14 days, showing an early stage of a dysplastic lesion. 1, Fused hallux and 2nd toe; 2, Blood effused into bleb; 3, Site at which effusion has taken place; 4, Bleb formed in the subcutaneous tissue of the dorsum of the foot; 5, Skin and subcutaneous tissue; 6, Tibia; 7, Os calcis; 8, Shaft of a metatarsal; 9, Third toe. (*After Bagg.*)

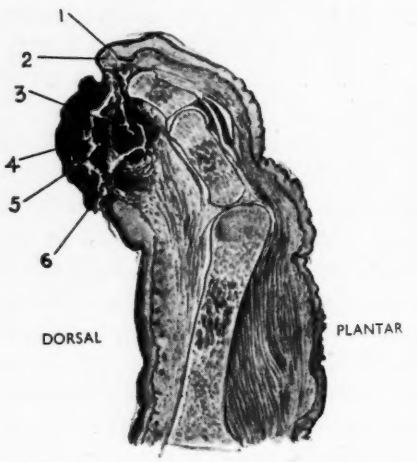

FIG. 116.—Section of the distal end of the foot of a mouse, one day old, showing later stage of a dysplastic lesion on the dorsal aspect. 1, Epithelium; 2, Remnant of distal phalanx with termination of cutaneous epithelium; 3, Hæmorrhage; 4, Necrotic tissue, including epithelium and red blood-corpuscles; 5, Fibrous tissue in state of reaction; 6, Epithelium dipping inwards at edge of necrotic tissues. (*After Bagg.*)

Now the lesion just described is similar in all respects to the dysplastic lesions of foot and digits described in the human fœtus by Dr. Streeter. There is, however, one important difference; the profuse formation of fibrous (amniotic) bands is absent. This difference must be attributed to a time factor. After the onset of the lesion the mouse has only a week to spend in the amniotic fluid before birth occurs, whereas the human fœtus is exposed to intra-uterine conditions for a period of 6 or 7 months. In mice, as in men, dysplastic lesions bring about intra-uterine amputations, distortion of feet and hands, and union of digits.

CONGENITAL CLUB-FOOT

The most common site of a dysplastic bleb in Dr. Bagg's strain of mice was the dorsal surface of the feet—both fore and hind. The effect of such lesions is to produce clubbing of the feet. In 432 mice born with deformity of the limbs, clubbing was present in 306 of them. Now, the lesion being usually on the dorsal side of the foot, the cicatrization which follows brings about a dorsiflexion of the foot—the deformity in man known as talipes calcaneus. In the Royal College of Surgeons Teratological Collection, there is a full time human fœtus (No. 434) with talipes calcaneus and necrotic areas on digits, showing the same lesions as in Dr. Bagg's mice; another example is figured by von Winckel.[2] Amongst the 306 instances of club-foot amongst mice 291 were of the talipes calcaneus form, and only 9 showed plantar flexion with inversion of the foot—talipes equinus varus; 6 others showed plantar flexion and distortion. Amongst human cases of club-foot the percentage of types of club-foot is reversed.

When one considers how different the human foot is from that of the mouse, and how far apart man and mouse are in the scale of evolution, a difference in the manifestations of the same disease need not astonish us. If a lesion of the nature described by Dr. Bagg were to form in the medial plantar aspect of the foot of an early human fœtus between inner malleolus and os calcis, then the pathological manifestations—the cicatrical contraction of all the ligamentous structures attached to calcaneus, internal malleolus, and navicular tuberosity which we find in cases of club-foot in man—would be produced. I have no longer access to early fœtal material in which the first manifestations of clubbing must appear, but I have no doubt that those who search will discover that what Dr. Bagg has found concerning the production of club-foot in mice is also true of club-foot in man. In brief, I am convinced that congenital club-foot in all its human forms is but one of the many ways in which Streeter's dysplasia becomes manifest in our fœtal bodies.

A high proportion of the dysplastic feet of mice shows division of a digit—usually the hallux or pollex. This is also so in human families which suffer from a dysplastic taint. If division of a digit is a dysplastic manifestation, then we have to ascribe the onset of these disorders to the time at which digits make their first appearance—namely in the 6th week.

SUMMARY

The object of this paper is to throw light on certain obscure congenital fissures and scars of the human face exemplified by two cases depicted in *Figs.* 84, 85. The conclusion reached is that these facial fissures are of the same nature as lesions described by Dr. George L. Streeter in the limbs of human fœtuses—lesions attended by the formation of fibrous rings and bands (so-called amniotic adhesions) and intra-uterine local necroses and amputations. The author proposes to distinguish the disorder which underlies such manifestations as a distinct clinical entity under the provisional name of 'Streeter's fœtal dysplasia' (dysplasia fœtalis).

Further light on fœtal dysplasia is thrown by two other embryological researches—one carried out in the laboratory of Prof. Florence Sabin by Miss Ellen Finley on the vascularization of the external coverings of the head; the other by Dr. N. W. Ingalls, who has described lesions along the mid-dorsal line of the human fœtus giving rise to certain cysts and scars which manifest dysplastic changes, often attended by the formation of 'amniotic adhesions'. The

author therefore regards congenital scars, and defects of the scalp, meningoceles (occipital and frontal), spina bifida, and the various degrees of anencephaly as manifestations of the same fœtal dysplastic disorder.

The various forms of dysplastic lesions of the face are then examined and exemplified, and the conclusion is drawn that all such lesions are caused by a local necrosis probably due to a circulatory failure which may be placental in origin. The failure occurs along marginal areas where capillary formation is in progress. The lesions become manifest at two stages of human development: (1) Towards the end of the first month; (2) Towards the end of the second month.

'Amniotic adhesions' are never formed by a failure in the separation of the amnion from the embryo, but are always produced by and from the fœtus—as a result of a dysplasia in fœtal tissues. They are the result, not the cause, of fœtal malformations. It would be well to substitute the name 'desmoplast' (δεσμός, a bond) for amniotic adhesions and to give the name desmoplasia to the process of their formation.

Dr. H. J. Bagg has bred a strain of mice which are particularly liable to dysplastic lesions of the feet. Members of this dysplastic strain are the descendants of parents which were subjected to brief doses of X rays, with the result that the parent germ plasm was damaged—a damage which has been transmitted in their descendants as far as the nineteeth generation. Dysplasia in Dr. Bagg's strain of mice —manifested by local necrosis, union of digits, and club-foot—has thus been made hereditary. The author is of opinion that the congenital lesions which Dr. Bagg observed in fœtal mice are of the same nature as those which Dr. Streeter observed in the extremities of human fœtuses. Although the matter requires further investigation, it seems highly probable that all forms of congenital club-foot in man are of dysplastic origin.

The author takes this opportunity of acknowledging the assistance given by the artist, Miss Barclay-Smith, who is responsible for the preparation of all the illustrations. Acknowledgements are also due to Dr. A. J. E. Cave and Mr. W. R. Le Fanu, of the Royal College of Surgeons of England.

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SACRO-ILIAC TUBERCULOSIS

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ALTHOUGH sacro-iliac tuberculosis is not a rare disease, it is unusual for an individual surgeon to have dealt with many cases; indeed the number of cases handled at any one hospital is rarely great. For this reason most writers on the subject have described only short series of cases, though Sisefsky (1933) who reported 66 cases is a notable exception. Generalizations based on relatively few observations are often misleading, and because there was reason to believe that this has been so with sacro-iliac tuberculosis, case records have been collected from a number of hospitals specializing in the treatment of joint tuberculosis to furnish sufficient material for reasonably conclusive analysis. Even so, a statistician might complain that the numbers are too small, but one has to be content with such material as is available, and the collection of cases to be reported is so far the most extensive on record.

MATERIAL

There are seven series of consecutive cases, and their origins are as follows:—

London County Council, St. Luke's Hospital, Lowestoft (for patients over 16 years of age)	68
London County Council, Queen Mary's Hospital for Children, Carshalton (for patients under 16 years of age)	3
Robert Jones and Agnes Hunt Orthopædic Hospital, Oswestry	42
Wingfield-Morris Orthopædic Hospital, Oxford	31
Lancashire County Council, Wrightington Hospital	16
Royal National Orthopædic Hospital, Country Branch, Stanmore	12
Harlow Wood Orthopædic Hospital	4
Total	176

In 5 cases the late histories were defective and these cases have been excluded from certain of the analyses.

Although it was impossible for us to examine all the cases reported, the value of the collection is unimpaired, as we have been fortunate in obtaining records from observers of the highest competence. We have also been at some pains to exclude certain cases in which the data (chiefly concerning diagnosis) appeared inconclusive, and in so doing may have worsened the picture of the disease, since most of the cases excluded pursued a favourable course; some of them were, perhaps, true examples of sacro-iliac tuberculosis. In certain respects, particularly in the late radiographic appearances, the records are defective; but after considering all the difficulties inseparable from follow-up work, we feel bound to pay tribute to the general excellence of the case notes and to the generosity of friends and colleagues who made them available to us.

CHIEF FEATURES OF THE DISEASE

The features of sacro-iliac tuberculosis are so well known that it is not proposed to describe all of them in full, but only where something new may be learned from the study of a large collection of cases.

Age Incidence (*Fig.* 117).—Sacro-iliac tuberculosis is essentially a disease of young adults, 80 per cent of cases occurring between the ages of 16 and 35.* This distinguishes it from all other forms of joint tuberculosis: the reason for this peculiarity is not known, and is worth investigating.

Sex.—There is little difference in the sex incidence. Of 176 cases, 85 (48·3 per cent) were males, and 91 (51·7 per cent) females.

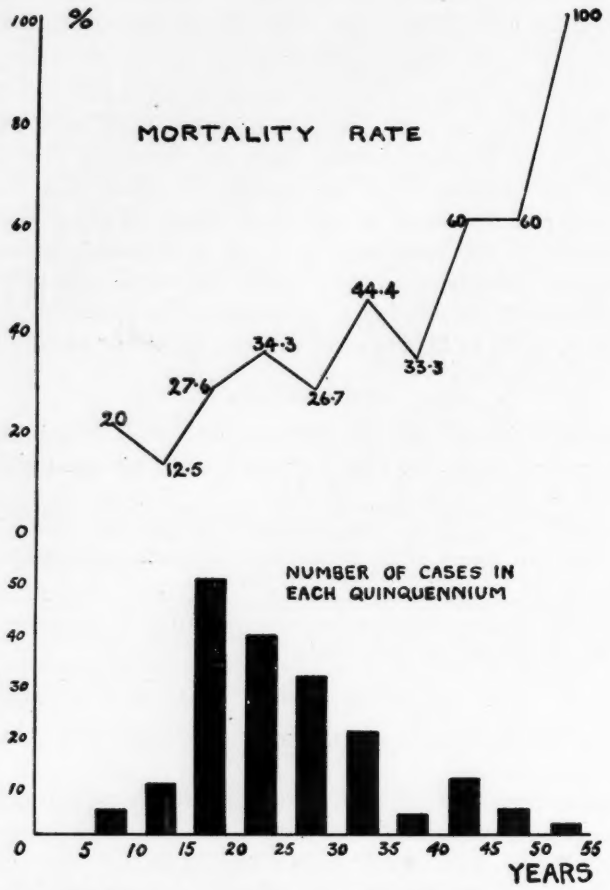


FIG. 117.—Chart showing age incidence of the disease and the mortality-rate in each quinquennium.

Pain.—Our records are hardly detailed enough to furnish information about the character and intensity of pain, nor, seeing that both depend so much on the patient's powers of description, would such information have much value. The distribution of the pain and its relation in time to the appearance of an abscess are of far greater importance.

The various *sites* of pain are shown in *Fig.* 118. Sciatica comes first, with pain in the hip and lumbar backache close seconds; pain over or in the sacro-iliac

* The fact that our cases were not all drawn from hospitals admitting children and adults alike is unimportant. London, for example, sent 68 adult cases to Lowestoft, but only 3 children to Carshalton during the same period.

joint is also common and pain in the buttock or thigh rare. The variations in distribution are due to the close relation between the joint and the lumbosacral plexus, and still more to the ability of an abscess arising from the joint to track in almost any direction and so involve one or more of the nerve-trunks in the vicinity.

Certain surgeons, particularly Elmslie, hold the view that in sacro-iliac tuberculosis pain and a manifest abscess are rarely present together. This is analogous to the rule, to which we have found few exceptions, that compression paraplegia in spinal tuberculosis and a superficial abscess do not occur together: this is so nearly constant that if an abscess becomes palpable in a patient suffering from paraplegia one can confidently expect the paralysis to disappear rapidly. The explanation is simple: a tightly confined abscess is able to compress the spinal cord, but one that is finding its way to the surface is undergoing spontaneous decompression and the tension in its walls falls. It is supposed that the same is

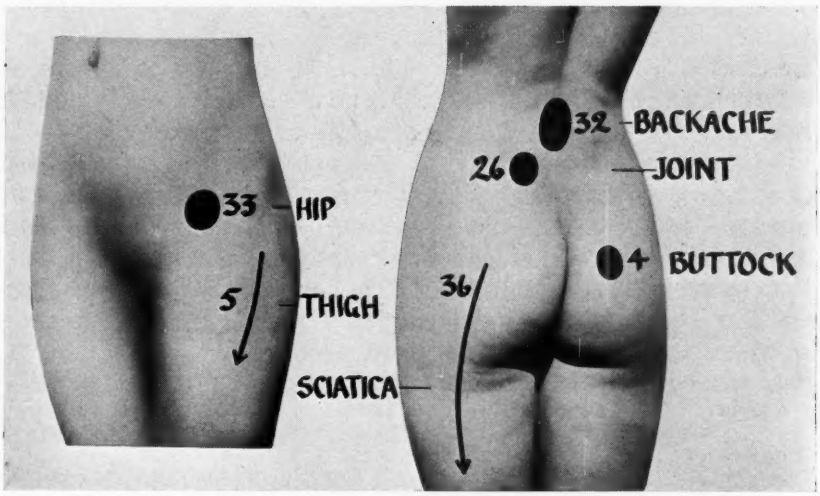


FIG. 118.—Diagram showing the chief sites of pain in sacro-iliac tuberculosis.

true in sacro-iliac tuberculosis: if the abscess is deep and confined it will produce pain in the distribution of any sensory or mixed nerves lying in or near its walls, presumably by venous congestion or actual stretching. If the abscess breaks through its tight wall and so makes its way to the surface or into the abdomen tension is reduced and the pain is relieved. If the abscess is at no time closely confined, and therefore not under tension, it will be palpable early in the course of the disease, and serious pain will not be experienced at any time. At the moment we are assuming (what later will be shown to be correct) that abscess formation is the rule in sacro-iliac tuberculosis.

To what extent is this clinical rule accurate? There was *absence of pain* in 21 cases, and in all but 1 an abscess was present and was the first indication that something was amiss. In the exceptional case the lesion was discovered radiographically in a patient known to be suffering from spinal tuberculosis.

In 44 cases *pain was a prominent symptom and at no time did an abscess appear.*

In 49 cases *pain was present at first, but ceased when an abscess appeared.*

In 43 cases pain and an abscess were present together.

It is clear that the first three groups of cases, 114 in all, support the clinical rule, whereas 43 do not.* We may say then that *in most cases pain due to sacro-iliac tuberculosis and an abscess do not go together.*† (*Table* 1.)

Table I.—Relation of Pain to Abscess Formation

	Pain and No Palpable Abscess	*Pain until Abscess Appeared*	*Pain and Abscess*
Backache	19	—	13
Pain in sacro-iliac joint	7	11	8
Pain in buttock	2	—	2
Sciatica	9	14	13
Pain in hip	6	22	5
Pain in thigh	1	2	2
Totals	44	49	43

Abscess Formation.—An abscess is a frequent though by no means invariable feature of sacro-iliac tuberculosis: an abscess (sometimes more than one) was present in 127 out of 176 cases (72 per cent). It almost always appeared early, and in 21 cases was the first sign of trouble. In the other 96 cases a visible or palpable swelling or a sinus was present either on or soon after admission to hospital. One cannot but suspect that there is generally a considerable interval between the onset of disease and the development of symptoms, for in no other joint—even the most accessible—does early abscess formation occur with such regularity. In other cases low back pain or sciatica was treated symptomatically for a considerable period before the patient was admitted to hospital—generally until it occurred to the clinician to ask for a radiograph or until the appearance of an abscess proclaimed the diagnosis. The term "early" is therefore used in relation to the time of admission to hospital rather than to the onset of the disease. In 5 cases an abscess appeared after six months in hospital, and in the other 5 after one year.

Table II.—Sites of Abscess Formation

Over sacro-iliac joint	64
Groin	17
Groin, gluteal region, and thigh (multilocular)	9
Gluteal	8
Lumbosacral	6
Iliac fossa	5
Thigh and trochanter	4
Over ilium	3
Lumbar	2
Over sacro-iliac joint and in iliac fossa	2
Gluteal and iliac fossa	1
Gluteal, iliac fossa, and femoral triangle (multilocular)	1
Internal	1
Position not stated	3
Abscess which might have arisen from coexisting spinal lesion	1
Total	127

* From analogy with other joints it should be possible to distinguish three types of pain: (1) Pain due to effusion into the joint; (2) Starting pains (occurring chiefly at night) during the stage of erosion of the joint surfaces; (3) Referred pain—already described in detail. It is conceivable that the first and second types may account for the cases that failed to conform to the general rule.

† Certain cases have been omitted either because a hip or spinal lesion was also present, or on account of atypical distribution of the pain. In 9 cases sciatica and backache were present together, and in 5 pain in the hip and backache.

The sites of appearance of abscesses are shown in *Table II*, and the more important of them are indicated in *Fig.* 119.

The rarity of abscess formation in the true pelvis is striking; in only 1 case could a swelling be felt per rectum.

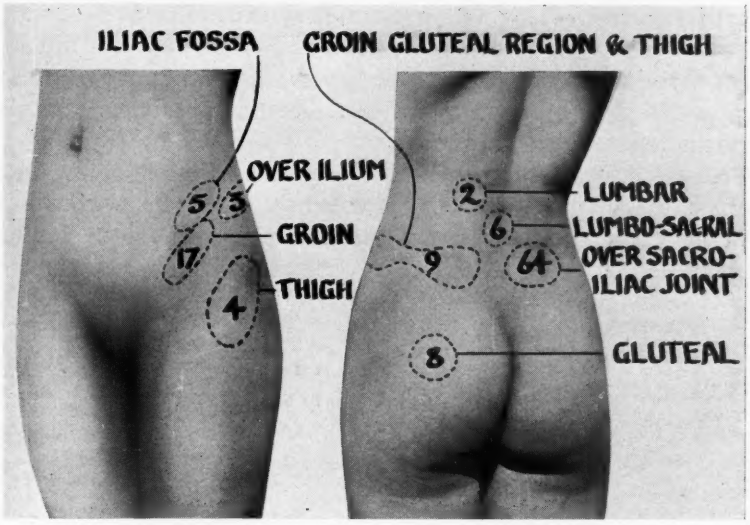


FIG. 119.—The chief sites of appearance of abscesses.

In 49 cases no abscess could be found, though adequate evidence in support of the diagnosis was otherwise available :—

Bacteriological or histological proof at operation or autopsy	9 cases
Associated lesions in which the diagnosis of tuberculosis was proved bacteriologically or histologically	20 cases
Diagnosis made on radiological grounds	20 cases
Total	49 cases

Undoubted radiological evidence of tuberculous disease was present in all cases.

Sinus Formation.—Sinuses were frequent. In 53 cases (30 per cent of 176) one or more sinuses complicated an apparently isolated joint lesion. In a further 21 cases with more than one manifest tuberculous lesion sinuses were also present over the sacro-iliac joint (12 per cent of 176), which brings the total incidence of sinuses to 42 per cent—74 cases in all. Accurate information about healing was obtained in 49 of the 53 cases of isolated lesions with sinuses. Sound healing occurred in 24 cases, and one or more sinuses persisted in 25. In the first group there was only 1 death, in the second there were 15—all traceable to secondary infection of the diseased joint. The importance of a sinus in its influence on the prognosis will be considered later.

Pathological Proof.—However obvious the diagnosis, no opportunity should be neglected of obtaining laboratory confirmation. One weakness of this collection of cases is that in addition to complete pathological proof being available in only 49 cases, there was the regrettable necessity of eliminating a number in which pathological evidence was essential to the diagnosis. One disease so hard to distinguish on clinical and radiological grounds alone is subacute pyogenic infection

of the joint or the adjoining bone, a condition in which abscess and sinus formation is not uncommon. Here the laboratory examination of material, repeated if necessary, is most essential, for although radiographically visible destruction of the sacro-iliac joint accompanied by a sinus is probably due to tuberculous disease, it *may* be due to pyogenic infection.

Bilateral Disease.—This was present in 5 cases. It is noteworthy that in four of them a spinal lesion was also found, and disease of the hip in the fifth. Two patients made a good recovery, 2 died, and 1 is gravely ill. In one case there was a considerable interval, eight years, between the appearance of the disease on one side and its development on the other.

Incidence of Associated Lesions.—Tuberculous lesions found in association with sacro-iliac disease are astonishingly frequent; they were present in more than one-third of the cases, and were sometimes multiple. It is to be emphasized that these associated lesions were all clinically obvious and it is possible that an even

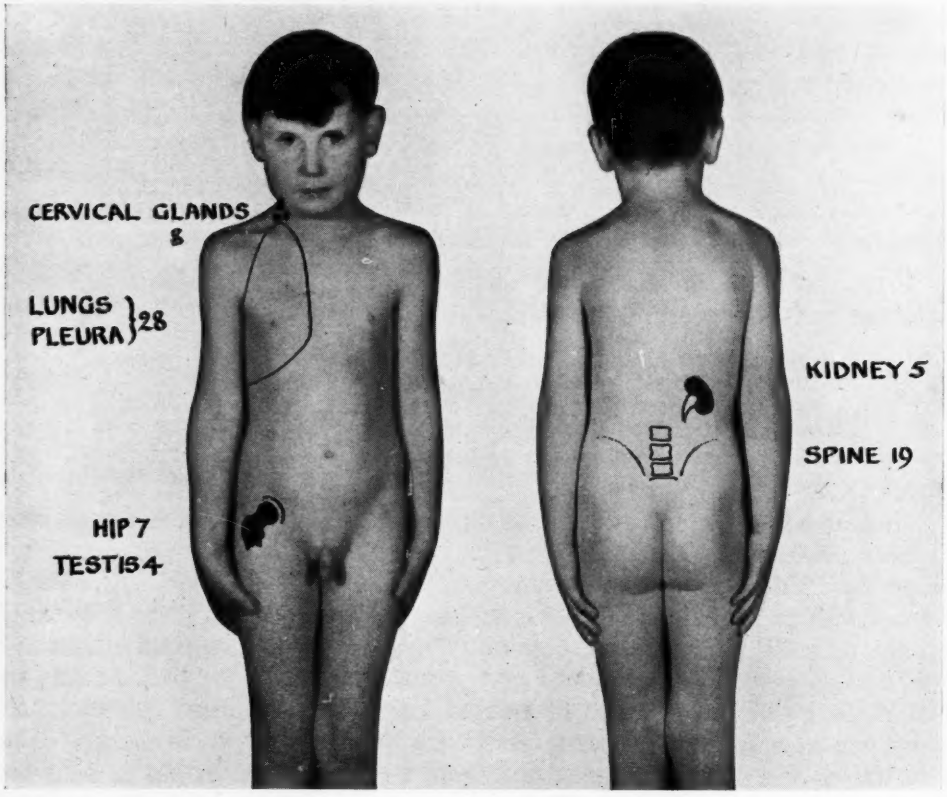


FIG. 120.—Sites of the more important associated lesions.

higher incidence would have been revealed by the routine investigation of the two notorious haunts of latent disease, the lungs and the kidneys. This finding is more than a demonstration of the roving propensities of the tubercle bacillus, once it has established itself in the body: it is an all-important factor in the prognosis, as will be shown later. (*Table III*; *Fig.* 120.)

As would be expected in a disease afflicting chiefly young adults, the lungs are commonly involved. Infection of skeletal structures is even more frequent, and in this category disease of the spine holds first place.

Table III.—INCIDENCE OF ASSOCIATED LESIONS
(*see also Fig.* 120)

Associated lesions (all obvious) in 63 cases (35·8 per cent)
Multiple associated lesions in 15 cases (8·5 per cent)
(included in the first figure)

Lungs and pleura	28
Larynx	1
Spine (*see Fig.* 005)	19
Hip	7
Skull	1
Clavicle	1
Sternum	1
Shoulder	1
Elbow	1
Wrist	1
Thumb (both joints)	1
Pubis	1
Knee	1
Ankle	1
Tarsus	1
Kidney	5
Testis	4
Fallopian tube	1
Breast	1
Skin (lupus)	2
Lymph-nodes: cervical	8
axillary	1
Ear	1
Conjunctiva	1
Palmar ganglion	1
Total associated lesions	91

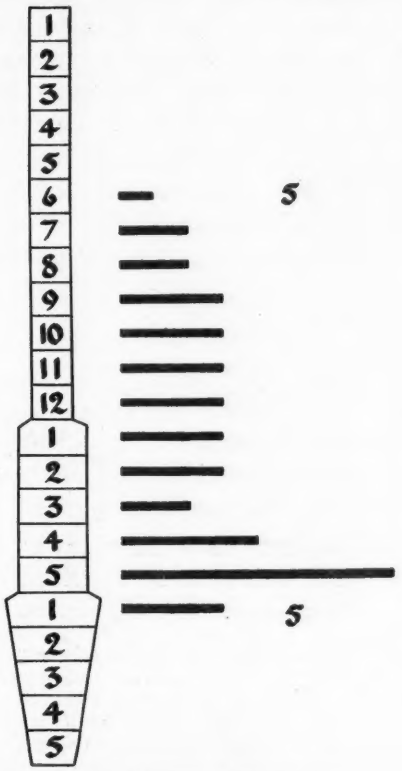


FIG. 121.—Diagram showing the distribution of associated spinal lesions in sacro-iliac tuberculosis.

It is interesting to note that the lumbosacral vertebræ (*Fig.* 121) are those most commonly affected, whereas the thoracolumbar region is the part specially favoured by uncomplicated disease of the spine. This curious finding, together with the fact that the hip comes second in the list of joints affected, lends colour to the suggestion that tuberculous bone infection is sometimes regional rather than strictly focal, the bacillary emboli reaching their destinations like pellets spreading out from a shot gun (the same phenomenon is often seen in the hip where foci appear more or less simultaneously in the acetabulum and the head and neck of the femur). It is always wise to look carefully at the lumbosacral region when examining the radiographs from a case of sacro-iliac tuberculosis.

Although the spine and the hip are the commonest sites of articular tuberculous disease, and it is therefore to be expected that they would head the list of associated joint lesions, this does not explain the frequency of involvement of the lumbosacral spine. There may, indeed, be a connecting link in addition to the one already described. The psoas muscle, so frequently involved in abscess formation, may act as a channel to convey infection between the spine, the sacro-iliac joint, and the hip: this is a possible route of spread that ought to be investigated.

Radiography.—The radiographic changes are entitled to the most detailed consideration, for chiefly on them is the diagnosis made.

Early Changes.—As in the hip and most other joints the lesion may be articular from the first, or para-articular. But it is not always easy to say how many cases begin with a lesion in the joint, and how many in the ilium or sacrum, for one cannot feel confident that the radiographic changes found on admission are necessarily those of the early disease: mention has already been made of the unusually 'early' appearance of abscesses, which suggests that the disease is generally well established when the patient first comes under observation.

Erosion of the joint surfaces was found as the first sign in most cases, and, as will be seen from *Table IV*, it was often advanced at the time of admission—still further evidence in favour of the disease having been present for a considerable time before the patient sought relief or before the correct diagnosis was made.

Table IV.—Erosion of Joint Surfaces

	Minimal Erosion	*Moderate Erosion*	*Extensive Erosion*	*Widespread Destruction*
Whole joint involved	34	45	22	2
Upper half alone	7	4	2	–
Central two-fourths alone	–	2	–	–
Lower half alone	8	24	3	–
Totals	49	75	27	2

153 cases

In 14 cases there was no trace of joint erosion, but there were other important signs that must be described in order to show that erosion of the joint surfaces is not an essential early feature of the disease:—

Iliac cavitation (abscess present)	3
Iliac cavitation (no abscess)	1
Sacral cavitation (abscess present)	1
Sacral rarefaction (abscess present)	1
Iliac and sacral cavitation (abscess present)	3
Iliac and sacral cavitation (without abscess but with spinal tuberculosis)	1
Intense rarefaction, increased joint space (abscess present)	1
Sclerosis, increased joint space (abscess present)	1
Sclerosis, diminished joint space (abscess present)	1
Sclerosis, normal joint space (abscess present)	1
Total	14 cases

Cavitation of bone, so common in the acetabulum and the upper end of the femur, is infrequent in sacro-iliac disease. There is nothing to suggest that many of the cases of erosion began as para-articular cavities, for in such cases the primary bone cavity would remain visible for a considerable period after the disease had invaded the joint proper.

Iliac cavitation (3 cases with early joint erosion also)	7
Sacral cavitation (5 cases with early joint erosion also)	6
Iliac and sacral cavitation (2 cases with early joint erosion also)	6
Total	19 cases

The radiographic appearances found on admission have been described in 176 of the 181 affected joints occurring in our collection of 176 cases. In 5 nothing useful could be learned either because the disease was obviously of considerable standing, or, in one case, because the patient was dying on admission. Erosion

of the joint—not invariably uniform—is the characteristic change, but it is possible for the joint surfaces to be preserved, at least in the early days, in which case one must rely on other radiographic signs to establish the diagnosis. Cavitation of the ilium or sacrum, or of both bones, is occasionally found and is inevitably followed by destruction of the joint surfaces.

A number of other changes are of minor importance, but not without interest. Gross *alteration of the joint space* was noted in 24 cases (13 per cent); in 12 it was increased, and in 12 diminished. In a few cases this increase or diminution was found in the absence of erosion of the joint surfaces. Increase is due presumably to ligamentous softening and effusion, and diminution to absorption of articular cartilage such as occurs so frequently in the spine (Seddon, 1935) at the stage when tuberculous granulation tissue has crept between the articular cartilage and the bone but has not as yet produced macroscopic bone changes.

Extreme rarefaction, a common finding in most forms of articular tuberculosis, is not often seen in sacro-iliac disease: it was present in only 13 cases (7·4 per cent). Early *sclerosis* was even less common: there were only 9 cases (5 per cent). *Sequestrum formation* was noted in 19 cases (11 per cent).

An uncommon but most significant finding is *luxation of the ilium upwards*. It is usually of minor degree, but may be gross and accompanied by a corresponding luxation at the symphysis pubis. It was found in 18 cases (10 per cent), and in all but one there was erosion of the joint surfaces; in one case there was cavitation of the sacrum without erosion of the joint. In no case was luxation found where the joint space was diminished. This condition corresponds with pathological subluxation elsewhere, and is an indication of widespread disease (*Fig.* 122).

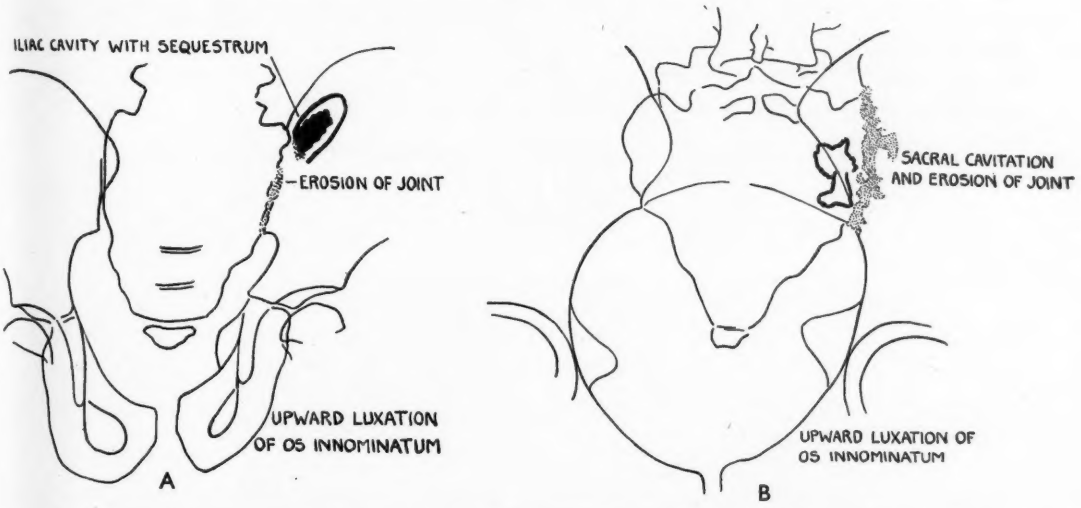


FIG. 122.—A, Tracing from radiograph showing gross upward luxation of the os innominatum and a cavity in the ilium containing a sequestrum; B, Tracings from radiograph showing a slight degree of upward luxation of the os innominatum; there is a cavity in the sacrum and widespread erosion of the joint.

Later Changes.—One of the most constant features of bone and joint tuberculosis is that, however early in the course of the disease treatment may be commenced, the destructive process usually advances for a time, sometimes for several months, before there is any sign of natural arrest. In most juvenile cases

this is clearly seen in serial radiographs. In adults, however, there is not always the same tendency to progressive destruction, so that its absence—in spine, hip, knee, and sacro-iliac joint alike—by no means excludes tuberculous disease. On the other hand, its presence confirms the diagnosis. It is not uncommon to find sacro-iliac tuberculosis following the same radiographic development as other tuberculous joint lesions—namely, rarefaction, bone destruction, progressive for a time, a period of stasis with no change in the radiographic picture, then gradual recalcification and a clearing of the bony trabeculæ, and finally fibrous or bony ankylosis. Serial radiographs are therefore of value; they must be taken, but the surgeon should not expect them always to show this orderly sequence of events as frequently as in lesions in children; he must be prepared to find cases in which there is little change after the first picture, apart from increasing calcification. This, again, is probably due to patients with sacro-iliac tuberculosis presenting themselves for treatment at a late stage—when the disease is fully developed and bone destruction maximal.

The traditional view that fibrous ankylosis is the usual result of conservative treatment prevails in regard to sacro-iliac tuberculosis. One could hardly expect anything else, for there is a constant shearing strain on the joint as inimical to bony fusion as the compressive stress in the vertebral column is favourable. But we must attempt to find out what is actually the case, for theoretical considerations are often misleading. Unfortunately, information on this point is very meagre, for if it is difficult to get patients to attend for follow-up examination, it is still more difficult to obtain late radiographs, and almost impossible to obtain them at regular intervals. There are only 15 cases (excluding those treated by operation) in which late radiographic information is reliable: but it is necessary to point out that hitherto nothing whatever has appeared in the literature in any way bearing on this question, which invests these fifteen cases with some importance. The incidence of fibrous and bony ankylosis is shown in *Table V*:—

Table V.—INCIDENCE OF FIBROUS AND BONY ANKYLOSIS (*see Fig.* 123)

Years after Discharge from Hospital	*Ankylosis still Fibrous*	*Bony Ankylosis first Observed*
1		2 cases*
2		1 case*
3		2 cases†
4	1 case*	1 case
5	1 case	
6	1 case*	3 cases†
7		1 case*
8		
9		
10	1 case	1 case*

* Indicates pathological proof that the lesion was tuberculous, or the presence of an associated tuberculous lesion.
† The same in one of the cases listed.

This is an unexpected discovery, and the proportion of cases (4 : 11) excludes any possible fortuitousness. In spite of the unfavourability of mechanical conditions, there is no doubt that bony fusion is the commonest end-result of tuberculosis of the sacro-iliac joint. A sinus was present in only 4 of the 11 cases, and it is therefore unwise to invoke secondary infection as the cause of the bony fusion. This finding has an important bearing on the question of operative fusion when we come to consider what, if anything, may be gained by operation.

A

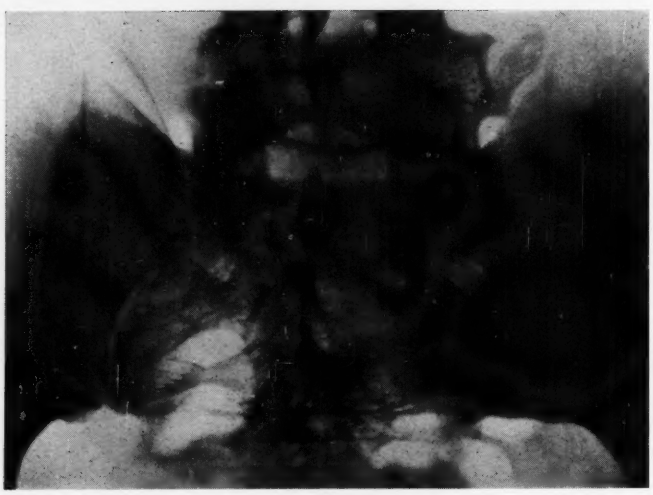
B

C

Continued on next page

D

E



F

FIG. 123.—A, Normal sacro-iliac joints; B, Erosion of right sacro-iliac joint and sclerosis of unknown origin on left; C, Erosion of right sacro-iliac joint with small sequestrum; D, Erosion of left sacro-iliac joint with cavitation of the sacrum; E, Erosion of right sacro-iliac joint and fibrous ankylosis of left; F, Bony fusion of left sacro-iliac joint following conservative treatment.

PROGNOSIS

It was this aspect of sacro-iliac tuberculosis more than any other that prompted us to undertake this survey. To spare the reader a tedious recital, we propose to omit reference to earlier views on prognosis since it is our desire rather to determine the outlook for the patient under present-day conditions. Most of us have been brought up to believe that the prognosis in sacro-iliac disease was poor, though no one seemed to know exactly how poor. Latterly, the favourable course of many cases has made surgeons more hopeful, and the current opinion now seems to be that the prognosis is not so gloomy.

A number of factors influence the prognosis and we propose to consider them in turn under six headings: (1) Age; (2) Life tables—(*a*) closed isolated lesions, (*b*) sinuses, (*c*) associated lesions; (3) Demographic factors; (4) Operative treatment; (5) Return to work; and (6) Pregnancy.

1. Age.—If we take as normal the mortality rate in the age period when the disease is commonest, i.e., 16–35 years, then it is evident (*see Fig.* 117) that the prognosis is better in children (6–15 years) and much worse after the age of 40.

2. Life Tables.—To construct *Table VI* we have used all the cases in which the histories were complete (171) and have calculated the mortality-rate* for each year (q_x), the survival rate for each year (p_x), and the cumulative survival rate (l_x)

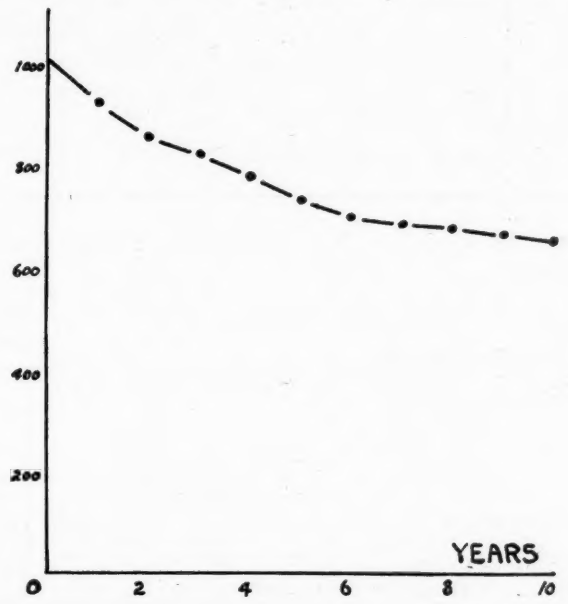


FIG. 124.—Life table for ten years, based on the study of 171 cases.

based on the conventional figure of 1000. The column l_x gives the theoretically correct number of survivors at the beginning of each year reckoned from the time of onset of the disease. Thus, of the 1000 starters 918 passed the one-year post—a mortality rate of 8·2 per cent. Only 732 survived for five years, a mortality-rate

* All deaths due to tuberculous disease or conditions arising from it, e.g., amyloid disease, have been included, but none due to intercurrent maladies such as carditis or simple pneumonia.

of 26·8 per cent over the five-year period; and 652 lived for ten years, a mortality-rate of 34·8 per cent for the decade. It is clear from *Fig.* 124 as well as from *Table VI* that the annual toll diminishes rapidly after the fifth year, and it is therefore safe to say that if a patient has managed to struggle through five years after the onset of the disease his ultimate chance of survival is excellent. Or, to coin an Irishism, the longer he lives, the more likely is he to continue to do so. This is similar to the conclusion reached by one of us (Seddon, 1938) in a parallel investigation into the prognosis of adult spinal tuberculosis.

Table VI.—LIFE TABLE

(10 years; 171 cases; reckoned from time of onset of disease)

YEAR OF THE DISEASE x	NO. OF CASES OBSERVED IN EACH YEAR	DEATHS OCCURRING IN EACH YEAR	ANNUAL SURVIVAL-RATE p_x	ANNUAL MORTALITY-RATE q_x	THEORETICAL NUMBER OF PATIENTS ALIVE AT BEGINNING OF EACH YEAR l_x
1	171	14	0·918	0·082	1000
2	153	11	0·928	0·072	918
3	139	6	0·957	0·043	852
4	127	6	0·953	0·047	815
5	121	7	0·942	0·058	777
6	101	5	0·950	0·050	732
					(26·8 per cent mortality)
7	79	1	0·987	0·013	695
8	68	1	0·985	0·015	686
9	56	1	0·982	0·018	679
10	44	1	0·977	0·023	667
					652
					(34·8 per cent mortality)

Here it is necessary to include a list of causes of death, and these are shown in *Table VII* below:—

Table VII.—CAUSES OF DEATH

Secondary infection	15	24
Secondary infection and amyloid disease	9	
Pulmonary disease		10
Tuberculous meningitis		5
Tuberculous nephritis		4
Generalized tuberculosis		3
Tuberculosis of the hip with sinuses		2
Tuberculous enteritis		1
Calculous nephritis		2
Other conditions		5
Exact cause of death unknown		5
Total		61 deaths

In certain cases where death was due to more than one cause, an attempt has been made to adjust the final figures accordingly; e.g., death due to pulmonary disease and sinuses with secondary infection was reckoned as pulmonary disease $\frac{1}{2}$, secondary infection $\frac{1}{2}$.

If it serves no other useful purpose, this list of causes of death shows at least one thing—that we are dealing with a lesion that is often only one manifestation of widespread infection. The life table, as it stands, is therefore of very limited

value, for it does not show to what extent the sacro-iliac infection *per se* was responsible for the fatalities. We have therefore endeavoured to divide the cases into groups in an attempt to discover to what extent the local disease was to blame.

An instructive investigation along the same lines has recently been published by Mather Cleveland (1939), and it is encouraging to find that his conclusions harmonize fairly well with ours. Unfortunately, the final figures cannot be compared because he did not construct life tables—which is the only accurate way of calculating mortality-rates in chronic disease.

The collection of 170 cases* has been divided into three clinical groups :—

a. Sacro-iliac disease appearing as an isolated manifestation and never giving rise to sinuses—a single closed lesion.

b. Sacro-iliac disease appearing as an isolated lesion, but with sinuses.

c. Sacro-iliac disease occurring with other manifestations of tuberculosis—excluding meningitis, which is regarded only as a terminal event. In this group further subdivision would have been desirable according to whether the sacro-iliac lesion appeared contemporaneously with those elsewhere and whether sinuses were present or not. But with such a small group of cases further subdivision is statistically unsound.

The life tables for these three groups of cases have not been carried beyond the sixth year, again because the numbers are small, and, more important, because the first five or six years have already been shown (*see Table VI*) to be the critical period ; lessons learned from this period are almost certain to be true throughout.

a. Closed Isolated Lesions (*Table VIII*).—The mortality-rate over the six-year period is only 9·7 per cent, which means that *the isolated lesion without sinuses is almost a benign condition.* At this point it is necessary to emphasize that

Table VIII.—Life Table for Cases of Isolated Sacro-iliac Disease Without Sinuses (6 years ; 56 cases)

Year of the Disease x	No. of Cases Observed in Each Year	Deaths Occurring in Each Year	Annual Survival-rate p_x	Annual Mortality-rate q_x	Theoretical Number of Patients Alive at Beginning of Each Year l_x
1	56	2	0·964	0·036	1000
2	52	1	0·981	0·019	964
3	51	0	1·000	0·000	946
4	47	1	0·979	0·021	946
5	40	0	1·000	0·000	926
6	40	1	0·975	0·025	926
					903
					(9·7 per cent mortality)

great pains were taken to exclude from this group all cases in which doubt could be thrown on the diagnosis. If anything we have been too harsh, and this mortality-rate of 9·7 per cent may be taken as a maximum figure.

b. Sinuses (*Table IX*).—

Here is a very different state of affairs—a mortality-rate of 24·5 per cent. It supports the traditional view about tuberculous abscesses, so well described by

* One of the 171 cases has been omitted because it was a little uncertain into which group it fell.

Calot, who said that " to open them is to open a door by which death too often will enter ". The presence of a sinus is a grave menace to life—and particularly when, as in the present case, radical surgical treatment (such as amputation of a limb) is inapplicable. Surgeons of an older generation used to warn their pupils

Table IX.—Life Table for Cases of Isolated Sacro-iliac Disease With Sinuses (6 years; 53 cases)

Year of the Disease x	No. of Cases Observed in Each Year	Deaths Occurring in Each Year	Annual Survival-rate p_x	Annual Mortality-rate q_x	Theoretical Number of Patients Alive at Beginning of Each Year l_x
1	53	2	0·962	0·038	1000
2	49	3	0·939	0·061	962
3	46	1	0·978	0·022	903
4	43	2	0·953	0·047	883
5	39	4	0·898	0·102	841
6	33	0	1·000	0·000	755
					755
					(24·5 per cent mortality)

that it was their business above all things to see that a sinus did not form; the means of saving life, by the proper treatment of abscesses, lay to a great extent in the surgeon's hands. In those days, when the indiscriminate opening of abscesses was common, and aseptic surgery a novelty, this was probably true; a sinus meant secondary infection and too often death. A glance at the table of causes of death (*Table VII*) shows that this is still a real danger. The surgeon is under the gravest responsibility to prevent secondary infection of a sinus, and the best preventive of all is to deal with abscesses by aspiration so that a sinus may never form.

A sinus in sacro-iliac tuberculosis raises the death-rate from 10 *to* 25 *per cent during the critical first six years of the disease.*

c. Associated Lesions (*Table X*).—This table gives us the appalling mortality rate of 55·2 per cent over the six-year period. *The presence of an associated lesion is of the gravest significance and, apart from advanced age, the most important factor*

Table X.—Life Table for Cases of Sacro-iliac Disease Occurring with Other Tuberculous Manifestations (6 years; 61 cases)

Year of the Disease x	No. of Cases Observed in Each Year	Deaths Occurring in Each Year	Annual Survival-rate p_x	Annual Mortality-rate q_x	Theoretical Number of Patients Alive at Beginning of Each Year l_x
1	61	11	0·820	0·180	1000
2	49	7	0·857	0·143	820
3	42	5	0·880	0·120	713
4	36	3	0·917	0·083	628
5	32	3	0·906	0·094	575
6	29	4	0·860	0·140	521
					448
					(55·2 per cent mortality)

in determining the prognosis. In some of the cases in this collection no deliberate search for silent lesions was made, and it is possible that in a few subclinical chest infection may have been overlooked. The lesson is obvious: in addition to the fullest clinical examination, radiography of the chest should never be neglected, and in the light of R. I. Harris's (1929) work we may find it wise to examine the urine periodically for tubercle bacilli (sometimes the only evidence of renal infection) even in the absence of suggestive symptoms. Neglect of radiography of the chest may lead to unpleasant surprises, as in the case of a patient in this series who was under the care of one of us (H. J. S.).

A girl of 13, Theresa N., was admitted to the Country Branch of the Royal National Orthopædic Hospital, Stanmore, with tuberculous disease of the left sacro-iliac joint. Apart from the local disease she appeared to be a healthy child: the chest was normal clinically and radiographic examination was thought to be unnecessary. She was treated conservatively in the usual way and appeared to make good progress apart from two slightly disturbing developments. Two months after admission a tuberculous abscess appeared in the left submaxillary region, and the blood sedimentation rate which had been 30 on admission rose during the following eight months to 52. The latter ought to have suggested the possibility of some other major focus developing, but it was not until a hæmoptysis occurred during her eighth month in hospital that the presence of extensive bilateral pulmonary disease was discovered. The girl began to cough and tubercle bacilli were found in the sputum. She died from pulmonary disease twenty-one months after admission.

It is possible that the lungs were normal on admission, but radiographs should have been taken at that time, and all the more when the blood sedimentation rate rose steadily instead of falling.

It is tempting to ask which type of associated lesion is most likely to influence the prognosis unfavourably, but a glance at *Table III* shows that one could hope to obtain reliable information in only two groups of cases—pulmonary disease and

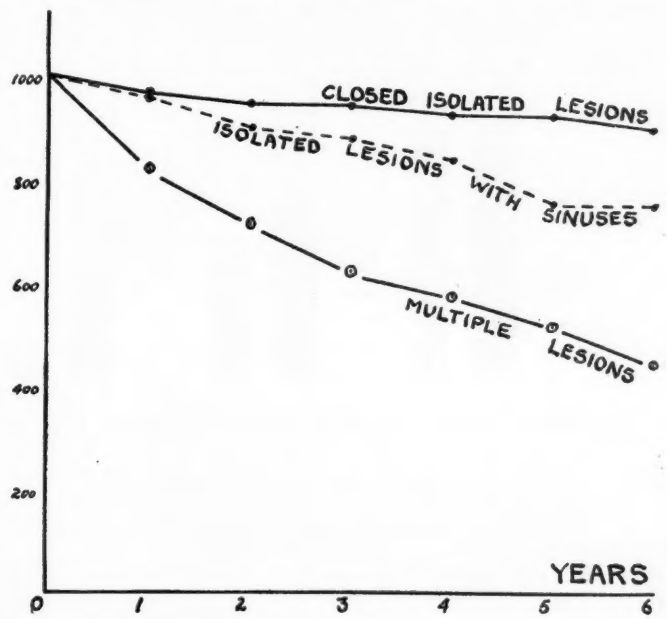


FIG. 125.—Composite life table showing: (1) The comparatively benign nature of closed isolated lesions; (2) The increase in mortality-rate due to sinuses; and (3) The disastrous influence of multiple lesions.

spinal disease—because the remaining lesions are all so infrequent. Where the lungs are involved the mortality-rate over the six-year period is a little under 50 per cent, and in the case of the spine nearer 60 per cent; but the groups of cases are so small that one cannot say that either shows any significant departure from the mortality-rate for the whole group, i.e., 55 per cent.

We have now arrived at a most important conclusion, which may explain differences in the experience of various observers. Sacro-iliac tuberculosis appearing as an isolated lesion without sinus formation is a comparatively benign disease—the mortality-rate over a six-year period is under 10 per cent. If sinuses are present, the mortality-rate jumps to 25 per cent, and if other tuberculous lesions are present, with or without sinuses, the outlook is very grave indeed, for more than half the patients are dead before six years have elapsed since the onset of the disease. These differences are shown very clearly in a graph made from the life tables (*Fig.* 125).

3. Demographic Factors.—In working through the clinical records, coming as they do from widely separated districts, one could not fail to be impressed by differences in the fate of patients in the several areas. Let us consider the Lancashire cases drawn exclusively from that county; the London County Council cases treated at Lowestoft—patients coming chiefly from the metropolitan area; and the Oxford cases drawn mainly from Oxfordshire, Buckinghamshire, and Berkshire. The Oswestry and Stanmore cases are excluded because many came from far afield.

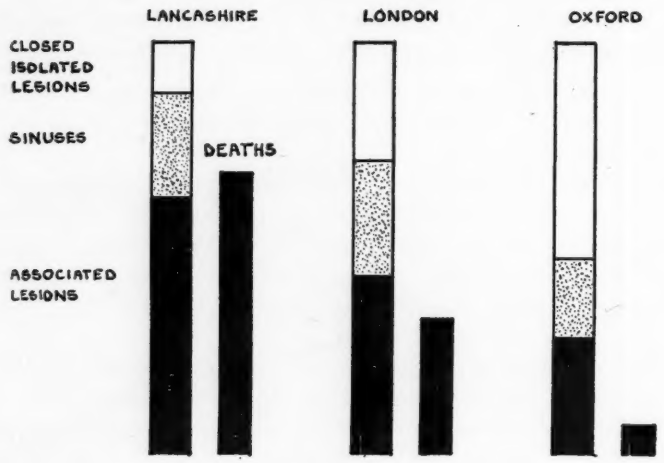


FIG. 126.—Diagram showing local differences in type of case and death-rate.

Table XI.—(*See also Fig.* 126)

	Lancashire	*London*	*Oxford*
Closed isolated lesions	2	19	14
Isolated lesions with sinuses	4	19	5
Associated lesions with or without sinuses	10	30	8
Total number of cases	16	68	27
Total deaths to date	11	23	2
Mortality-rate (approximate)	69 per cent	33 per cent	7·4 per cent

At first sight one would say that sacro-iliac tuberculosis is not the same disease in Lancashire as it is in Oxford. In the former not only is it nearly ten times more lethal, but associated lesions are far more frequent—which in itself goes far to explain the higher mortality-rate. Consideration of figures such as these shows the futility of comparing " results of treatment " at this or that centre when it is so patent that the disease under consideration behaves differently in different districts. (*Table XI*; *Fig.* 126.)

What is the explanation? There is, so far as we know, no magic at Oxford unknown to Wrightington or Lowestoft. It is true that most of the Oxford cases were treated by arthrodesis, but would such treatment have saved the Lancashire patients from dying of meningeal, pulmonary, or renal tuberculosis? It is equally inconceivable that such glaring differences could be explained by local differences in hospital treatment. Nevertheless it is possible, even probable, that a patient's fate might somehow be influenced by what happens *before* he reaches hospital, and the two obvious factors to be considered are: (1) Delay in arriving at the correct diagnosis; (2) Improper treatment of an abscess before admission, leading to sinus formation and secondary infection.

We have investigated these factors in all cases where full information was available, and the conclusions are as follows (*Table XII*; *Fig.* 127):—

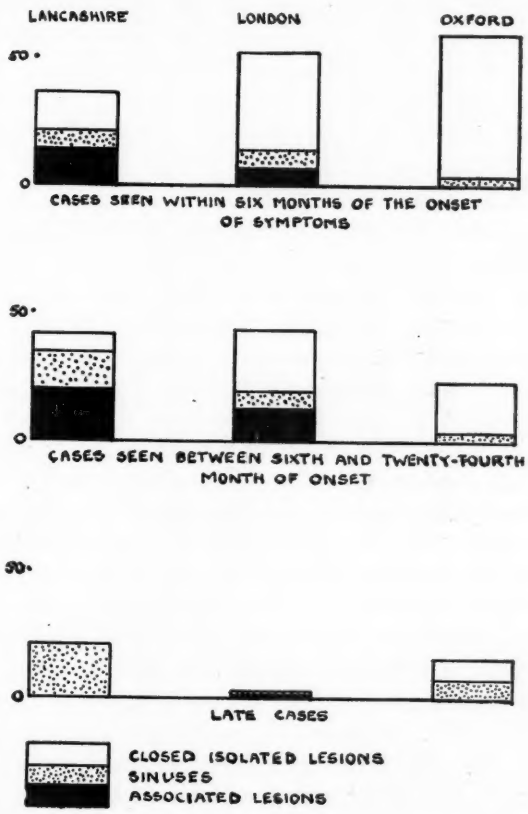


FIG. 127.—An attempt to show at what stage, and in what condition, patients were admitted for treatment in Lancashire, London, and Oxford.

Table XII.—The State of Patients on Admission to Hospital

	Lancashire	*London*	*Oxford*
Patients admitted within 6 months of the onset of symptoms	5	33	15
Sinuses present on admission	1	7	1
Associated lesions present on or developing after admission	2	7	0
Deaths	4	9	3
Patients admitted between 6 and 24 months after the onset of symptoms	6	27	6
Sinuses present on admission	2	4	1
Associated lesions present on or developing after admission	3	8	0
Deaths	5	9	0
Patients admitted late—neglected cases	3	2	4
Sinuses present on admission	3	2	2
Associated lesions present on or developing after admission	0	1	0
Deaths	1	2	2

In two important aspects Oxford was superior to Lancashire and London; more cases were seen and admitted during the early months of the disease: and the incidence of sinus formation before admission was commendably low. The Wingfield-Morris Orthopædic Hospital is the centre of an extensive and well-organized clinic system, and there is no question that practitioners and outlying hospitals in Oxfordshire, Buckinghamshire, and Berkshire refer orthopædic cases for treatment at a relatively early stage and there is little meddlesome treatment of abscesses. There is no comparable system in Lancashire or London, and to us it is reasonable to connect the relatively benign type of cases dealt with at Oxford with the efficiency of the net that catches them. But this is not the whole story: in Lancashire and London alike there was a high incidence of associated lesions even in cases seen early, and this is a factor beyond the control of the best clinical organization. There was therefore an element of good fortune as well as good management at Oxford.

4. Operations.—As will be seen later, these local variations make it almost impossible for us to compare the results of operative treatment at one centre with those of conservative treatment at another. Little is known about the influence of operation on the prognosis, though the problem, tackled in the right way, should be capable of solution.

5. Return to Work.—This aspect of the prognosis is next in importance to life itself, and is often uppermost in the mind of the patient. We have been able to follow over a considerable period the progress of almost all patients who left hospital—some for more than ten years—and in a substantial majority the details of their employment are known. In order to clarify the conclusions nothing short of full work has been considered; a few incorrigible gentlemen of leisure and neurotic women have been listed as "unfit for work", although this was untrue so far as their physical condition was concerned. The prognosis is therefore rather better than the figures would suggest, though how much better it is impossible to say. (*Table XIII*; *Fig.* 128.)

During the first year, and more particularly in the first six months after leaving hospital, most patients were unfit for full work or perhaps afraid to begin. During the second to the fifth years strength and confidence returned, a certain amount of light work was undertaken, and the majority returned to full labour. After the

fifth year almost every patient was hard at work, and suffering no permanent disability.

There was a little chronic invalidism due to sinuses, though certain patients with persistent sinuses suffered only the inconvenience of a daily dressing. But

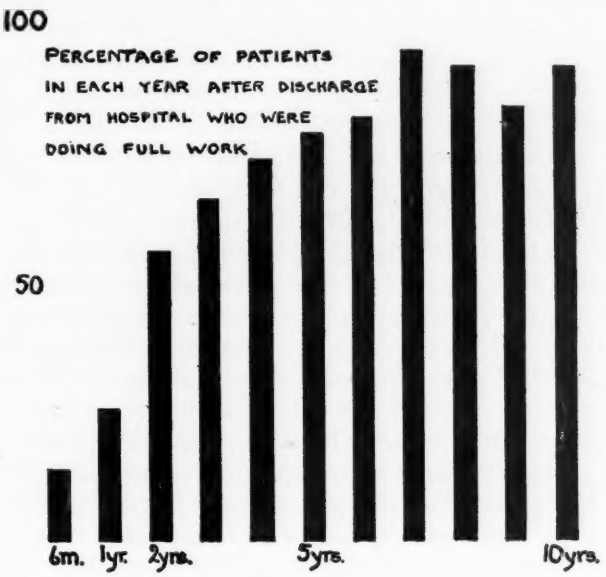


FIG. 128.—Percentage of patients in each year after discharge from hospital who were doing full work.

Table XIII.—RETURN TO WORK

Years after Discharge from Hospital	*Number of Patients under Observation in Each Year*	*Number Back at Work*	*Percentage Back at Work*
½ }	91	{ 13	14
1 }		{ 24	26
2	89	51	57
3	81	54	67
4	73	55	75
5	56	45	80
6	42	35	83
7	29	28	96
8	27	25	93
9	20	17	85
10	14	13	93

all things considered we may say that *after recovery from sacro-iliac tuberculosis there is little or no permanent incapacity either for work or sport.* The performance of certain patients (which need not be detailed here) was indeed remarkable, several of them having returned to work as miners.

6. Pregnancy.—To what extent does pregnancy precipitate the onset of sacro-iliac tuberculosis, and what, if any, is its influence on a joint that is supposed to have healed?

Only 4 cases furnish evidence in answer to the first question. In two the onset of the disease occurred during pregnancy, and in the other two the onset followed soon after delivery. These numbers are too small to be of value.

Ten patients became pregnant after having suffered from sacro-iliac tuberculosis. The details of 9 of them are as follows :—

1. Normal pregnancy 3 years after discharge.
2. Normal pregnancy 8½ years after discharge.
3. Normal pregnancy 9 years after discharge.
4. Normal pregnancies 1 and 7 years after discharge.
5. Normal pregnancies 1 and 6 years after discharge.
6. Normal pregnancies 2 and 6 years after discharge.
7. Normal pregnancies 2 and 7 years after discharge.
8. One miscarriage 5 years, and normal pregnancies 7 and 9 years after discharge.
9. One pregnancy 2½ years after discharge; terminated on the advice of the surgeon in charge of the case, but not because there was any suggestion of relapse.

In none of these 9 cases has there been any recrudescence of the disease. In the tenth case, a woman of 24, who had had severe sacro-iliac tuberculosis with sinuses, pregnancy occurred five years after discharge from hospital. Disease in the joint again became active, and she returned to hospital for one year. She lived for two and a half years after leaving hospital the second time and then died of amyloid disease.

It is safe to say that if a woman with apparently well-healed sacro-iliac tuberculosis becomes pregnant, *there is every prospect of her going to term and through labour without showing reactivation of the disease.* In the ordinary course of events there is no need to advise termination of the pregnancy. This accords with the opinion of Myers (1891) in regard to pregnancy and tuberculous disease of the spine: even in his time it was found that pregnancy rarely had an adverse effect on a lesion that appeared to be well healed.

TREATMENT

There is such a measure of agreement on the principles governing non-operative treatment that we propose to confine this discussion to the controversy that still continues about the merits of operative treatment as opposed to conservatism. This controversy embraces almost all major joints affected by tuberculosis, including the sacro-iliac joint.

In the nineteenth century half-hearted conservatism was the rule; there was a scarcity of beds, treatment was generally of short duration, and the guiding principles imperfectly understood. Towards the end of the century, as a result of the radical operative treatment of malignant disease becoming established, it was natural for enterprising surgeons to apply similar methods to joint tuberculosis. They argued that if the bony or articular focus could be eradicated good hygiene would look after the hidden glandular focus that started all the trouble without the necessity of confining the patient to hospital for a long period. This was the era of eradication, excision, and erasion, and the results of such treatment are too well known to require detailed description; it is sufficient to say that not only did such operative heroics generally fail to arrest the local disease, but because they often involved destruction of epiphyses in growing patients they resulted in gross deformity and the most appalling shortening of limbs.

The present century brought a more enlightened conservatism, and a realization that in order to obtain good results time must be no object. Local and general

treatment were to be continued until the patient himself had overcome the disease, and a sound ankylosis, generally fibrous, had been obtained.

It had long been known that bony ankylosis was the only entirely satisfactory end-result, a result unfortunately rare—though sometimes obtained at some risk to the patient where secondary infection of a sinus had occurred. This was the justification for excision of accessible joints such as the knee, and in adults at any rate this most desirable bony fusion was obtained with some regularity by operative means. But excision was limited to a few accessible joints. From 1911 onwards the extra-articular method of arthrodesis associated with the names of Albee and Hibbs extended the scope of fusion operations immensely, and it was thought that here, at last, was a rational alternative to the tedious, expensive, and sometimes unreliable conservative methods. It was claimed that extra-articular fusion was in itself a conservative procedure, for it did not attempt eradication of the disease, but provided complete rest for the lesion which could then heal undisturbed by movement; it also guaranteed bony fusion from the start. This new gospel, apparently so rational in its conception, was received with such enthusiasm that many surgeons never stopped to question its premisses, but devoted all their energies to devising operative methods that would give sound extra-articular fusion in a substantial majority of cases. In certain places faith in the method became so well established that the fusion operation was endowed with almost mystical powers of healing,* and a patient with a sound operative fusion was said to be 'cured'.

Unfortunately, the new venture brought its disappointments. The disease, particularly in children, often continued to advance in spite of the fusion, and although certain surgeons, notably those of the Hibbs school, stoutly maintained that advancing disease was an indication of imperfect fusion and could be remedied by improvements in technique—or alternatively, that even if bone destruction did continue after operative fusion the extent of the damage was less than would have been the case had no fusion been performed—it came to be realized that *the best field for the operation was not in curing the disease, but, as a terminal procedure, in converting an unsound fibrous ankylosis into a stable bony fusion.* Throughout this whole controversy only one surgeon, Leo Mayer, of New York (1938), has had the courage and patience to apply precise methods to the obtaining of a solution. Over a period of ten years he treated alternate cases of spinal disease in children by early operative fusion (the others being treated conservatively) with results that very clearly favoured conservative methods. Similar, though less exact, investigations by McKee (1937) and Seddon (1938) have yielded much the same results in adult tuberculosis of the spine.

These trends of thought have influenced the treatment of sacro-iliac tuberculosis. It seemed the ideal joint for operative fusion; mechanical conditions appeared hardly favourable to sound spontaneous union and one sacrificed no useful range of movement by operating on a joint that moved so little anyway. Also the disease is commonest in adults, and there is still the widely held belief that, whatever the shortcomings of the fusion operation in children, it is the method of choice in adults.

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* *See* Dr. Benjamin Farrell's remarks in the discussion following the reading of Dr. Leo Mayer's paper (*Jour. Amer. Med. Assoc.*, 1938, **110**, 483).

There are several ingenious methods of fusion, as well as the older excision of Picqué (1891). All that is necessary is to determine if fusion is worth while. This has never been done; the advocates of operation have presupposed its superiority and have been content to report short and sometimes impressive series of successes. Some patients died, generally from tuberculosis elsewhere, but this was merely a misfortune and no reflection on the operation or the surgeon, who had simply done his best. With a regularity that seems to be an inherent characteristic of our calling, surgeons have divided themselves into conservatives and radicals, and have tried to reach a conclusion by studying the results of one type of treatment, and one alone. The treatment of alternate cases by operation has hardly been considered, much less practised. Undoubtedly there are many occasions when such cold-blooded inquiry is inadmissible where human life is concerned, but in the treatment of joint tuberculosis no such considerations need disturb the most sensitive conscience, for we know that operative treatment at its best is rarely a life-saving measure, and, to a critical mind, not invariably certain to cut down the duration of treatment. Another factor that makes the alternate-case method so supremely important is the possibility of local variations in the character of a disease. We have already seen that striking differences exist in the different groups of cases in our collection; in Lancashire the percentage mortality was of the order of 70, in London 33, in Oxford 7. We have no right to compare the excellent results of operative treatment at Oxford with the moderate results of conservative treatment at Lowestoft, or the discouraging results at Wrightington. The difference may have been in the local character of the disease. Such conclusions as can be drawn from our collection of cases are therefore tentative, and the final answer is still a long way off.

Types of Operation.—There are two chief types of operation: a more or less thorough excision of the focus of disease, indicated particularly in the presence of sepsis or sequestra; and arthrodesis, which aims at the speedy production of bony fusion either across the joint or by means of an extra-articular bridge of bone.

In this collection of cases there were 54 operations performed on 49 patients. All were carried out by experienced surgeons, and there was one death attributable to the operation.

Excision and Eradication.—Few surgeons will contest that in certain circumstances, namely, the presence of secondary infection or large sequestra with sinus formation, the preliminary treatment of a tuberculous focus in bone is the same as that required in chronic pyogenic osteomyelitis. One aims at obliterating or 'saucerizing' a cavity which could not possibly heal naturally, or removing a mass of dead bone which if left would cause sinuses to persist indefinitely. The actual technique is of little importance provided access is adequate, and it is not always necessary to perform the radical though rather elaborate operation described by Picqué (1891).

Operations of this nature are required only in unfavourable cases, and it is not surprising to find that the results are not altogether cheering. Too often the surgeon is faced with a virulent type of infection, perhaps with sepsis as a complication.

Although there were only 6 successes in this series of 12 cases, it must be remembered that in every instance the surgeon was fighting a rearguard action, and it is reasonable to believe that where healing occurred it was largely due to the operative intervention. There can be no doubt that these operations were necessary.

RESULTS OF EXCISION AND ERADICATION

No.	TYPE OF OPERATION	INDICATION FOR OPERATION	RESULT
1	Picqué	Widespread disease	Died: renal and sacro-iliac tuberculosis
2	Picqué	Long-standing disease	No improvement
3	Eradication	Persistent sinuses	Success
4	Eradication	Rapidly advancing disease	Died: sinuses and pulmonary disease
5	Eradication	Persistent sinuses	One small sinus: otherwise satisfactory
6	Eradication	Widespread disease	Success
7	Eradication	Abscess and sequestra	Success
8	Eradication	Three operations for widespread disease	Never healed: Died after 4 years—? cause
9	Eradication	Two operations for sequestra	Persistent sinus: died after 2 years—? cause
10	Eradication	Sinus and sequestra	Success
11	Eradication	Sinuses	Success
12	Eradication	Sequestra	Sinus persisted: died after 2 years: secondary infection

In 3 of the more favourable cases eradication of the focus was combined with a fusion operation—there being no secondary infection. In two cases bone chips were used, and an iliac graft in the third. The results were not encouraging:—

RESULTS OF ERADICATION PLUS FUSION

No.	TYPE OF OPERATION	INDICATION FOR OPERATION	RESULT
1	Sequestrectomy; iliac graft	Sequestra	Joint healed; died pulmonary disease
2	Eradication; bone chips	Long-standing disease	Sinus; no bony fusion
3	Eradication; bone chips	Long-standing disease	Much pain; no bony fusion

Fusion Operations.—Verrall's operation was the most popular: it was performed on 21 occasions. Smith-Petersen's followed with 14 cases, and Richard's operation was performed in a solitary case. On theoretical grounds Smith-Petersen's operation is less sound than Verrall's, for the graft is taken from the ilium in the immediate neighbourhood of the lesion and is countersunk across the joint and therefore lies in the centre of the infected area. However, the clinical results were if anything better than those of Verrall's operation.

In all these cases the operation was undertaken with a view to shortening the duration of hospital treatment, and in most it was carried out after only a short stay in hospital. In four-fifths of the cases the operation was performed when the patient had been in hospital for not more than six months, the duration of pre-operative treatment in all the straightforward cases being from three to seven months. This early resort to operation is less open to criticism than in the case of the hip or the spine, for as we have already shown (pp. 196, 200) there are reasons to believe that few cases of sacro-iliac tuberculosis are admitted before the disease

is well advanced and sometimes almost reaching the stage of quiescence. Nevertheless, in cases which on admission appear to have early lesions it is probably wise to delay operation until it is certain that the disease has passed beyond the active phase.

RESULTS OF VERRALL'S OPERATION

No.	Complications	Fusion of Graft	Fusion across Joint	Clinical Result
1	—	+	?	Satisfactory
2	—	+	?	Satisfactory
3	—	Disease continued		Failure
4	Abscess after 1 year	+	?	Satisfactory
5	—	+	+ after 4½ yr.	Satisfactory
6	—	+	Sequestrum present	Satisfactory
7	—	+	?	Satisfactory
8	Abscess after 2 years	?	?	Satisfactory
9	—	?	?	Apparently healed; but painful
10	—	+	?	Satisfactory
11	Abscess	?	?	Sinus
12	—	?	?	Satisfactory
13	—	+	?	Satisfactory
14	Slight paralysis of foot	+	+ after 6½ yr.	Satisfactory
15	—	+	+ after 3 yr.	Satisfactory
16	—	?	?	Satisfactory
17	—	} Follow-up incomplete		
18	—			
19	—	+	+	Died of renal tuberculosis
20	Wound infection			Died 4 months later; septicæmia
21	—	?	?	Satisfactory

RESULTS OF SMITH-PETERSEN'S OPERATION

No.	Complications	Fusion of Graft	Fusion across Joint	Clinical Result
1	—	+	+ after 2½ yr.	Satisfactory
2	—	+	+ after 5 yr.	Satisfactory
3	—	+	+ after 2½ yr.	Satisfactory
4	—	+	—	Satisfactory
5	—	—	—	Satisfactory
6	—	—	—	Satisfactory
7	—	Follow-up incomplete		
8	—	?	?	Satisfactory
9	—	?	?	Satisfactory
10	—	?	?	Satisfactory
11	—	?	?	Satisfactory
12	—	?	?	Satisfactory
13	—	?	?	Satisfactory
14	—	?	?	Died of generalized tuberculosis after 4 yr.

RESULT OF RICHARD'S OPERATION

The patient died after 1 year; ? cause

With one exception (Verrall: *Case* 20) the operation was without mortality: in this case a wound infection followed the operation and the patient died of septicæmia four months later.

Other complications were infrequent, and in no case serious. The appearance of an abscess has been listed as a complication, but it might have occurred in any case, and is simply an indication that operative fusion is no more capable of arresting disease in the sacro-iliac joint then in the spine or hip.

Fusion of the graft was observed in 11 cases treated by Verrall's method and in 4 treated by Smith-Petersen's method. Complete figures are not available because the follow-up examinations did not invariably include serial radiographic studies of the affected joint. This was a singularly unfortunate omission since we are interested not only in following the fate of the graft, but even more in its ability to promote bony ankylosis of the joint—which is the chief aim of the operation. In only 6 cases was certain evidence of *bony fusion* available, and in none did it appear any earlier than in cases where it followed conservative treatment. We have therefore no means of checking Smith-Petersen's opinion (which there is no reason to doubt, but which ought to be confirmed) that bony fusion soon follows operation in the majority of cases. It also remains to be proved that a bony ankylosis stands up better to the wear and tear of everyday life than does a fibrous ankylosis. It *should* do, but our observations show that patients with sound fibrous ankylosis have little to fear, and almost all are doing their full work without restraint.

The *clinical results* were excellent, which may explain why a number of patients (many of whom were seen after discharge in rural clinics remote from facilities for radiographic examination) were not submitted to routine radiographic examination—valuable though this would have been.

Complete recovery			26 (72 per cent)
Failure :—			
a. Apparently healed but persistent pain	1	4	
b. Persistent sinus	1		
c. Disease continued	1		
d. Died: wound infection	1		
Failure unconnected with the operation—			
a. Died: renal tuberculosis	1	3	
b. Died: generalized tuberculosis after 4 years	1		
c. Died after 1 year (? cause)	1		
Follow-up incomplete		3	
			36 (100 per cent)

It has been claimed that the *time spent in hospital is reduced* by operative fusion—an important consideration. Two groups of cases are offered for comparison: recovery under conservative treatment; and recovery following operative fusion.

Cases treated conservatively—average stay	18·8 months
Cases treated by operative fusion—average stay	17·3 months

The difference is too small to be significant.

But before considering these figures as a fair index of the influence of operative treatment on clinical results and the length of stay in hospital it is necessary to inquire into the type of case dealt with by operation. It has already been shown

that so far as prognosis is concerned there are three very distinct clinical types of sacro-iliac tuberculosis (p. 210): (1) A closed isolated lesion, in which the prognosis is excellent; (2) An isolated lesion with a sinus, where the outlook is not so good; and (3) A lesion with or without a sinus, but accompanied by a tuberculous lesion elsewhere—a type in which the six-year mortality-rate is more than 50 per cent.

Now the ratio between these groups for the whole series is almost unity, i.e., 56 : 53 : 61, whereas the cases treated by operation were as follows:—

Closed isolated lesions	23
Isolated lesions with sinuses	5
Associated lesions present	7

It is not often that one is able to demonstrate surgical judgement or selection so clearly statistically, but here is good evidence that the surgeons concerned chose most of their cases for operation from the very group that we now know shows the most favourable results whatever the method of treatment. Furthermore, most of the cases (22 out of 36) treated by operative fusion were under the care of Professor Girdlestone and, although the results are excellent, it must be recalled that sacro-iliac tuberculosis as seen at the Wingfield-Morris Hospital is a much less serious disease than in other areas from which cases were drawn (*see* p. 210, *Table XI*)—and it is possible, though not certain, that his cases would have done almost equally well under conservative treatment.

One reluctantly reaches the conclusion that in this series of cases there is no sure basis for comparing the merits of operative and conservative treatment. One thing is certain—that *there is little to suggest that operation is as valuable as its exponents believe*; in America and this country alike there is need for further comparative work which must be carried out on lines that will yield data susceptible of accurate analysis.

SUMMARY

A collection of 176 cases of sacro-iliac tuberculosis is reviewed in detail.

Chief Features of the Disease.—

1. It is a disease of young adults, and there are three distinct clinical types: (*a*) An isolated lesion without sinuses—33 per cent of cases; (*b*) An isolated lesion with sinuses—31 per cent of cases; (*c*) A lesion, with or without sinuses, but found in association with tuberculous lesions elsewhere—36 per cent of cases.

2. Abscess formation is exceedingly frequent, occurring in 72 per cent of cases, and usually to the exclusion of pain, the pain subsiding when the abscess appears.

3. Sinus formation is also frequent, occurring in 42 per cent of cases.

4. The commonest sites for associated lesions are the lungs and joints, particularly the lumbar spine.

5. The severity of the disease and the incidence of associated lesions vary from one part of the country to another.

6. Erosion of the joint surfaces is the commonest radiographic finding: para-articular cavitation of bone is not infrequent.

Prognosis.—

1. Prognosis as to life, as shown by life tables, depends chiefly on the clinical type of the disease. The mortality-rate over a six-year period is as follows: Closed isolated lesions, 10 per cent; Isolated lesions with sinuses, 25 per cent; Lesions

associated with tuberculous foci elsewhere, 55 per cent. After six years the mortality-rate becomes almost negligible.

2. Recovery generally means that the patient will be able to return to full work, and relapse is rare. A certain amount of chronic invalidism is due to sinuses.

3. Pregnancy does not cause relapse, provided that the disease has healed soundly.

4. Bony ankylosis is probably the usual end-result of conservative treatment, even in the absence of secondary infection.

Treatment.—

1. The principles of conservative treatment are well known and should be followed in every case.

2. Operative fusion of the joint may be beneficial, but its merits have not yet been clearly demonstrated.

3. The average period of hospital treatment is eighteen months, and is not materially altered by operation.

Without the generous assistance of many friends and colleagues this paper could not have been written.

For access to case records at Carshalton and Lowestoft we are indebted to Sir Frederick Menzies, late Medical Officer of Health for the County of London, to Dr. H. O. West, Medical Superintendent, Queen Mary's Hospital for Children, Carshalton, and to Dr. H. J. A. Colvin, Medical Superintendent, St. Luke's Hospital, Lowestoft.

To Dr. G. Lissant Cox, Central Tuberculosis Officer for the County of Lancaster, and to Dr. Alan Pask, Medical Superintendent, Wrightington Hospital, near Wigan, we are indebted for the records of cases treated at that Hospital: also to the surgical staff at the Robert Jones and Agnes Hunt Orthopædic Hospital, Oswestry, for the records of their cases, and particularly to Mr. G. T. Partridge who made us a free gift of the records that formed the basis of an excellent review of sacro-iliac tuberculosis presented by him at a meeting of the British Orthopædic Association at Oswestry in May, 1939.

Professor Girdlestone placed all his cases at our disposal and gave us a detailed report on the late condition in almost every instance. We are grateful also to the surgical staff of the Royal National Orthopædic Hospital, who allowed us to regard their cases as our own. We have also to thank Mr. S. A. S. Malkin and Mr. V. J. M. Taylor for the records of four cases treated at Harlow Wood.

Our friend Mr. W. Herschell has been most helpful in the preparation of diagrams and statistical tables.

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THE NATURE AND CAUSE OF SWELLING OF THE UPPER LIMB AFTER RADICAL MASTECTOMY

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THIS paper records an investigation into the nature and cause of swelling of the upper limb after radical mastectomy. The group of cases investigated has been previously reported by us (Devenish and Jessop, 1937) from the clinical aspect. These cases are characterized by the development of swelling of the upper limb after radical mastectomy in the absence of demonstrable recurrence. Observations were also made on patients recently subjected to this operation, and on some other patients. It will be convenient first to describe the methods employed and the results obtained in the present investigation, and subsequently to discuss their significance.

NATURE OF THE SWELLING

Evidence of the nature of the swelling of the upper limb was obtained by three different methods—namely, clinical observation, radiographic appearance, and dye injection of the skin lymphatics.

Clinical Observations.—Clinical observations showed that the swelling was always of two kinds, one pitting, the other not pitting. The usual assumption that pitting indicates the presence of œdema fluid was supported by observations on the shrinkage which occurred with continuous suspension of the limb.

It was observed that on elevation of the limb shrinkage occurred most rapidly in the areas which pitted on pressure. Those areas which had been hard and non-pitting became, after varying periods of elevation, soft and pitted on pressure, but in no case was there a complete restoration to normal. There was always some residual swelling of a brawny indurated consistency, this being most marked in those parts of the limb swollen for the longest time. A distinction must be made between the hard non-pitting swelling present before treatment and the residual brawny indurated swelling left after long elevation of the limb. The former we believe to be due mainly to a high degree of tension of œdema fluid in the tissues, but the nature of the latter is obscure. It may partly be due to fibrosis of the subcutaneous tissue as the result of long-standing œdema, but the evidence available is inconclusive.

Measurement of Shrinkage on Elevation.—The amount of shrinkage that followed elevation of the limb was estimated by displacement measurements of the volume of the limb. An arm bath filled to the brim with water was placed in a larger empty bath. The limb was then immersed in the full arm bath, and the volume of the water displaced into the outer bath measured. This was done three times and the average taken. Care was taken to see that the hand and forearm were fully immersed to the same skin mark each time.

Results: Quantitative measurements of the volume of the normal and of the swollen limbs were made to determine the increase in volume of the swollen limb and the degree of shrinkage that followed elevation. Only two measurements of the swollen limb were made, namely, before the beginning of elevation and at its end. Owing to the trying nature of this treatment, it was difficult to get the patients to tolerate it for long periods, and the figures therefore do not necessarily represent the maximum degree of shrinkage possible. The four patients thus treated were all women who had moderate or severe swelling of the affected limb, and in none was there any evidence of a recurrence of carcinoma. The essentials of their histories and the results obtained are given in *Tables I* and *II*.

Table I.—HISTORIES OF CASES INVESTIGATED

CASE	AGE IN YEARS	YEARS SINCE OPERATION	SWELLING			ATTACKS OF INFLAMMATION IN SWOLLEN LIMB
			Time of Onset	Duration	Degree	
R. P.	61	9	Seven years after operation	2 years	Moderate	None
D. U.	50	7	One month after operation	7 years	Severe	Two attacks, first 3 years ago, second 1 year ago
K. S.	68	18	Sixteen years after operation	2 years	Severe	None
A. C.	52	8	Immediately after operation	8 years	Severe	Two or three attacks in the last ten years

Table II.—MEASUREMENTS OBTAINED IN CASES IN *Table I*

CASE	VOLUME OF NORMAL LIMB	VOLUME OF AFFECTED LIMB (Initial)	PERCENTAGE INCREASE IN VOLUME OF AFFECTED LIMB	DAYS SUSPENDED	VOLUME OF AFFECTED LIMB (Final)	LOSS OF VOLUME OF AFFECTED LIMB	LOSS OF VOLUME EXPRESSED AS PERCENTAGE OF NORMAL LIMB
R. P.	2160 c.c.	2660 c.c.	+ 23 per cent	19	2600 c.c.	60 c.c.	2·8 per cent
D. U.	2950 c.c.	4210 c.c.	+ 43 per cent	24	3700 c.c.	510 c.c.	17·3 per cent
K. S.	1500 c.c.	2530 c.c.	+ 69 per cent	19	2170 c.c.	360 c.c.	24·0 per cent
*A. C.	1950 c.c.	4190 c.c.	+ 115 per cent	13	3000 c.c.	1190 c.c.	61·0 per cent

* We wish to draw attention to the fact that in our previous paper this patient was, owing to a clerical error, incorrectly reported as having lost 3 litres from the swollen limb after elevation.

If the increase in volume of the swollen limb and the change in its volume which occurs following elevation are expressed as percentages of the volume of the normal limb and this relationship is plotted as a graph (*Fig.* 129) it will be seen that there is nearly a straight line relationship between the initial percentage increase and the percentage loss of volume on elevation. If the increase in volume in the swollen limbs were due mainly to fibrosis, it would be difficult to explain this relationship, as elevation of the limb for comparatively short periods (14 to 24 days) is unlikely to produce a loss of fibrous tissue.

If, on the other hand, the swelling is due largely to an accumulation of fluid in the tissues, then it is reasonable to expect that the volume lost as the result of elevation will be directly proportional to the degree of swelling present initially. This is, in fact, the case. Together with the actual volume lost, which in the case of the patient A. C. was rather less than a quarter of the total bulk of the swollen limb, this provided strong evidence that most of the swelling is due to an accumulation of fluid in the tissues.

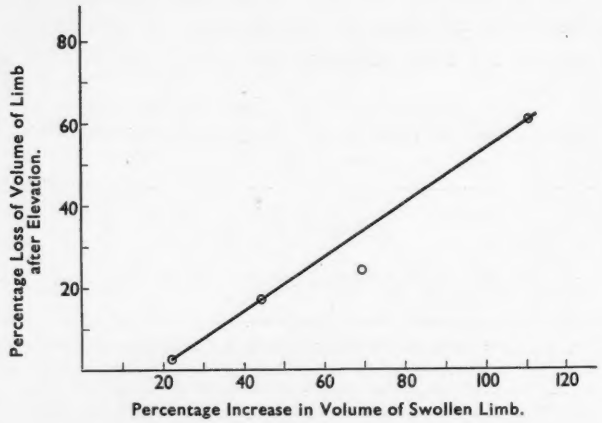

FIG. 129.—Graph showing the relation between initial increase in volume of the swollen limb and loss of volume on elevation, expressed as a percentage of the volume of the normal limb.

Protein Content of the Oedema Fluid.—In two patients small quantities of œdema fluid from the swollen arm were obtained by inserting a Southey's tube. In both the fluid was slightly blood-stained, but in calculating the total protein of the œdema fluid, the error due to introduction of blood-cells and plasma was corrected by red-cell counts of the œdema fluid and of the blood of the patients. The two patients were A. B. and D. U., and the total plasma protein and protein in the œdema fluid were 5·75 g. per cent and 2·99 g. per cent, and 6·21 g. per cent and 2·17 g. per cent respectively. The protein estimations were made by the method of Wu and Ling as modified by Fuenberg (1929).

Radiographic Appearances.—An attempt was made to get further information of the nature of the swelling by taking radiographs of the soft parts.

Method: Comparable radiographs of both forearms were taken in four cases. In three cases the swelling had been present 3, 3, and 11 years respectively. The swelling was of moderate degree in two cases and severe in the third. In the fourth case swelling was noticed on the fifth day after radical mastectomy, and the radiograph was taken on the twenty-fourth day. The swelling in this case was of moderate degree.

Results: The radiographs of the swollen forearm in each case showed considerable change in the appearance of the subcutaneous tissues from that of the normal side (*Fig.* 130). The latter showed a moderately clear strip containing a faint reticular pattern of somewhat greater density. The margin of the deep fascia, as seen in radiographic section, was clearly defined and smooth, and the deep surface of the skin was just discernible. In the former the depth of the subcutaneous tissues was much increased. They were occupied by a coarse reticular pattern of

considerable density, enclosing less opaque areas. The margin of the deep fascia was in places obscured by this pattern, whilst in some areas a smooth, denser uniform shadow was present at the cutaneous margin.

We were at first inclined to attribute the appearance of a coarse, dense reticulum in the subcutaneous tissues to the presence of fibrosis. The occurrence of a residual, non-pitting, brawny swelling would then be explicable as being due to this growth of fibrous tissue. But we abandoned this idea when in the absence of inflammation identical changes were found within 24 days of operation. It is very unlikely that extensive fibrosis would have taken place throughout the subcutaneous tissues in so short a period, especially in the absence of obvious inflammation. An alternative explanation is that the clearer spaces in the reticulum are due to the fluid in the tissue spaces, and that the denser areas represent the normal solid subcutaneous tissues.

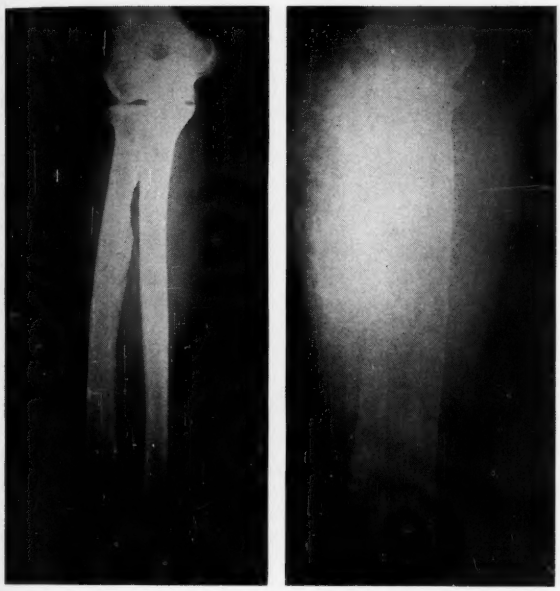

FIG. 130.—Radiograph showing the change in appearance of the soft parts of the swollen limb.

Dye Injection of Skin Lymphatics.—The intracutaneous injection of a highly diffusible dye according to the method of Hudack and McMaster was also employed to investigate the nature of the swelling. Details of the technique are given on p. 228.

The injections showed that the cutaneous lymphatics in the swollen limb were dilated and more permeable than those of the normal side. No evidence of fibrosis in the swollen limbs was obtained. Attacks of inflammation were described by five out of ten patients, but in no case was inflammation followed by a permanent increase in the size of the limb, nor was it present at the onset of the swelling. It is possible, however, that such attacks may leave some fibrosis in the subcutaneous tissues.

CAUSE OF THE SWELLING

If the swelling of the limb is nearly all due to œdema of non-inflammatory origin, it is clear that, in the absence of any general cause, such œdema must be produced either by a preponderance of filtration of plasma from the capillaries or by a diminished rate of lymphatic drainage or by a combination of these two processes.

VENOUS PRESSURE

As there is no evidence of interference with the arterial blood-supply of the affected limb, and as such attacks of inflammation as occur do so after the swelling is already established, it seems likely that a preponderance of filtration from the

capillaries can be produced only by a pressure in the veins of the affected limb higher than that of the normal. Such an increased venous pressure might be produced by compression of the veins in the axillary scar, though it is extremely unlikely that venous obstruction alone will produce œdema: the classical experiments of Cohnheim (1889) having demonstrated that ligation of veins usually fails to produce œdema—experiments which have often been repeated on man, in the operating theatre, and with the same results. Drury and Jones (1927–9), however, have shown that in normal subjects increased venous pressure of a sufficient height and maintained for long enough will produce palpable œdema. But as œdema will not develop unless the rate of loss of fluid from the capillaries exceeds the rate of absorption, and as in these experiments the increased venous pressure was produced by an encircling pneumatic cuff which necessarily compressed the lymphatics, and presumably stopped the lymph-flow, it is clear that a higher venous pressure than they found effective, i.e., 60 mm. Hg for 2½ hours, would be necessary to produce œdema in the same time in the presence of normally acting lymphatics. Krogh et al. (1932) estimate that "to produce a preponderance of filtration it would be necessary to raise venous pressure until the average difference between capillary pressure and colloid osmotic pressure in the absorbing area was slightly less than the average difference between these two pressures in the filtering area . . . this should occur at slightly over 25 mm. water pressure". Though this pressure in the presence of normally acting lymphatics would be unlikely to produce œdema, yet with lymphatic obstruction such a slightly raised or possibly even a normal venous pressure acting over a long time might produce œdema.

Venous pressure in the normal and affected limbs was investigated in the following manner:—

a. **Clinical Observations.**—If the pressure in the veins of the affected limb is raised, these veins should on inspection be fuller than those of the normal limb. Such comparisons must be made under conditions which will exclude changes in the calibre of the vessels due to unequal warming or unequal activity of the limbs. The hands and forearms were inspected, therefore, after they had been exposed to room temperature for twenty minutes, and had been at rest for this period.

The comparison is sometimes impossible when there is much swelling of the limb, but in those cases in which the œdema was moderate and those in which the œdema had been sufficiently reduced by elevation, there was no difference in the size of the veins. Those on the dorsum of the hand were usually chosen for comparison. In the presence of a normal arterial blood-supply a raised venous pressure can only be produced by obstruction to the venous outflow from the limb, which will result in delay in emptying of the affected veins. This may be made obvious by the simultaneous passive elevation of both limbs, the level at which the vein collapses being taken as a convenient end point. Care must be taken, as described above, that the veins are in a comparable state before the observations are made.

The observations were carried out as follows: The patient was sitting quietly in bed, with both arms exposed and at rest for twenty minutes. Each arm was then supported and elevated by one observer simultaneously, and the state of the veins on the forearm and on the dorsum of the hand watched. That this test will demonstrate the presence of venous obstruction is shown by the following observation.

J. S., male, aged 72 years. Exploratory operation on the neck for carcinoma of the cervical œsophagus was followed by suppuration of the wound due to a temporary œsophageal fistula, and swelling of the left upper limb from thrombosis of the left axillary, subclavian, and jugular veins. The swelling of the limb subsided as the result of continuous elevation of the limb. The observations on the veins were made nine and thirty-eight days after the onset of the thrombosis, when the limb was still moderately swollen. On examination, the veins of the left forearm were fuller than those of the right; and on elevation, while the veins of the right forearm collapsed when the wrist was at the level of the angle of Louis, those of the left did not begin to drop till the wrist was 10 cm. vertically above this point, and were not quite collapsed after being held in this position for five minutes.

Dye tests by the technique described later were made when the limb was swollen, and repeated eight days later when the swelling had almost completely disappeared.

In the affected limb, where there was no flow of lymph towards the axilla when the limb was swollen, eight days later, when the œdema had subsided, the lymph-flow, though slower than on the normal side, had re-appeared. The significance of these observations will be discussed later.

The elevation test of venous pressure was applied to a number of patients who had undergone radical mastectomy. Some of these suffered from swelling of the upper limb; in none was any difference observed in the rate of emptying. We may conclude that in this group of cases there was no clinical evidence of obstruction or raised pressure in the veins of the affected area.

b. **Direct Venous Pressure Measurements.**—These were made on the veins of the normal and abnormal arms simultaneously. They were carried out in three patients; one with severe swelling of the arm after radical mastectomy, one without swelling of the arm after the same operation, and one without swelling of the arm after an extensive axillary vein thrombosis some years previously. The method was adapted from Moritz and von Tabra (1910).

Though in all three patients the venous pressure on the affected side was slightly higher than on the normal side, yet this difference in pressures (a maximum of 3·9 cm. of saline) was too small to be significant. Further, in both cases which had been subjected to radical mastectomy, there was no difference in the behaviour of the affected limbs, though one had a swollen arm and the other had not. The measurements of venous pressure in the third case show that even though there had been an extensive block, efficient anastomotic channels had been provided.

In the light of these observations and experiments we may conclude that in none of the patients investigated was there any evidence of venous obstruction. Such rise of venous pressure as occurs during exercise (Hooker, 1911) can be a contributing factor in œdema formation only in the presence of lymphatic obstruction.

LYMPHATIC DRAINAGE

Dye Injection of Skin Lymphatics.—If the above is correct, then the only mechanism left to account for the swelling is some gross diminution in the rate of lymphatic drainage. This, however, is only negative evidence and needs confirmation, which may be sought by measuring the rates of lymph-flow in the affected and the normal limbs. For this measurement the method of Hudack and McMaster (1933) has been used. It consists essentially in the injection of small quantities of a highly diffusible dye into the skin lymphatics, observation of the resulting pattern and the rate of streamer formation yielding information as to the state of the lymphatic plexus and the rate of movement of the lymph-stream.

Method: An 11 per cent aqueous solution of Patent Blue V,* which is isotonic at this concentration, was used, and the technique employed closely followed that described by Hudack and McMaster. The syringe was a 1-c.c. metal-tipped tuberculin syringe graduated in hundredths, and the needle was of stainless steel, 0·30 mm. external diameter and 0·14–0·15 mm. internal diameter. The solution of dye was freshly prepared and sterilized by autoclaving, and the needle and syringe were sterilized in alcohol and carefully dried before use.

The experiments were carried out with the patients sitting quietly in bed or in some cases with the patients sitting at a table and with the forearms supported before them. In the latter, a period of 30 minutes' rest was allowed before the experiments were started. The forearms were comfortably supported so as to be nearly horizontal with the elbows flexed.

The skin was sterilized by pouring a little 70 per cent alcohol over the sites selected, which were the volar surfaces of both forearms about 10–15 cm. distal to the medial epicondyle of the humerus. From 0·02 to 0·04 c.c. of the 11 per cent solution of Patent Blue V was injected into each, and the injection was made "exceedingly superficially" into the stretched skin. The injection time, the subsequent appearance and behaviour of the injection pattern, and the rate of streamer formation were noted. Before a camera was available records were made by tracing the patterns and the streamers on a celluloid film held flat against the limb, using the dye itself as ink. Such tracings were subsequently copied on to paper. Later photographs were taken as a routine measure at 15, 30, 45, and 60 seconds during the injection, and at 1, 3, 5, and 10 minutes after the injection, as well as 'long shots', which included the whole of both limbs.

The camera used was a Leitz Leica, fitted with the Summar *f*2 lens. One type of film was used throughout the work, viz., Agfa ISS Panchromatic, although any other type of film of equal speed could be used. All films were developed by time and temperature method in a tank, and the developer used was a combined metol meritol fine-grain developer made according to Johnson's formula. The lighting unit was a portable double-unit stand, fitted with Photoflood lamps in conical-shaped aluminium reflectors (Kodak). For the pictures of the whole limbs, both lights were used at a distance of about 3 ft. from the patient. Lens aperture was *f*6·3 and exposure $\frac{1}{20}$ second, with the Leica deep red filter on the lens (Fnooy).

For the injection work the Leitz 1 : 1 reproduction attachment was fitted on the camera. For the photographs of the actual injection, the base plate of the reproduction device was removed and the legs of the stand rested lightly on the limb. This was done for convenience in handling the syringe. As soon as the needle was withdrawn the base plate was replaced and all subsequent photographs made through the 1 : 1 aperture. The lens stop was *f*3·2 and exposure $\frac{1}{8}$ second with the red filter on the lens. For illumination, only one of the Photoflood lamps was used, concentrating the brightest circle of light given from it by the conical reflector on to the opening in the base plate in the reproduction attachment. The lamp was about 1 ft. from the camera.

Results: Observations were made on one normal female and on ten patients who had been subjected to radical mastectomy. In the case of the normal control, the subject was a young woman, aged 26 years. Into each forearm 0·03 c.c. of

* The dye was manufactured by I. G. Farbenindustrie Aktiengesellschaft, and supplied by Messrs. I. H. Dyestuffs Ltd., 142–146, Old Street, London, E.C.1.

dye was injected. There was very little difference in the appearance of the injection patterns and in the rate of streamer formation, though slightly larger streamers appeared in the left than in the right arm in the same time.

In two patients with grossly swollen arms, similar and quite characteristic phenomena were observed. There was a striking difference in the injection patterns produced in the normal and in the affected arms. In the former the dye either remained as an intracuticular vesicle or produced a small localized network of injected lymphatics. In the latter an extensive and complicated network covering an area much larger than that found in any normal arm was produced. In addition, the dye entered the skin of the swollen arm more easily and rapidly and the injection was consequently more easily carried out. Soon after the injection had been completed, streamer formation was visible on the normal side, but none on the affected side during the whole period of observation, i.e., 30 minutes. Diffusion of the dye, as shown by blurring of the sharp initial pattern, was more rapid on the swollen limb, and in addition, the skin of the forearm gradually became discoloured by a fine bluish network, giving the impression that the dye was seeping through a sponge-like mass.

The details of one of these experiments are illustrated in *Figs.* 131–135 (*Case* K. P.).

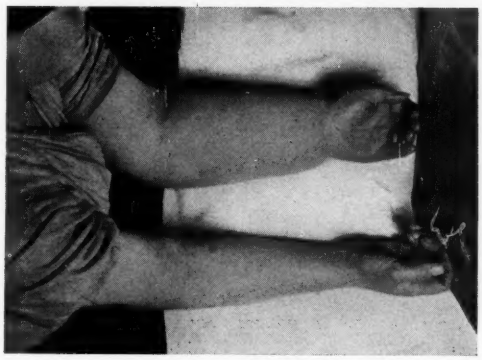

FIG. 131.—*Case* K. P. Showing severe swelling of the left forearm.

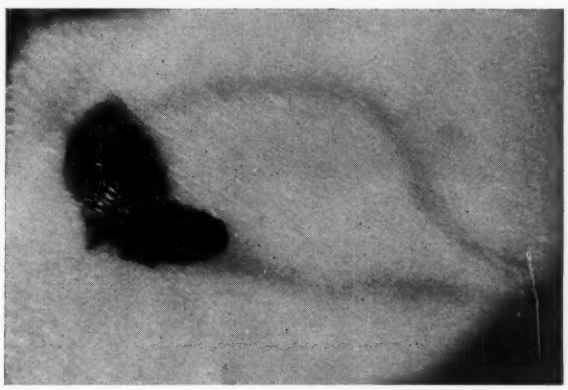

FIG. 132.—Same case, showing pattern 17 minutes after injection into normal forearm.

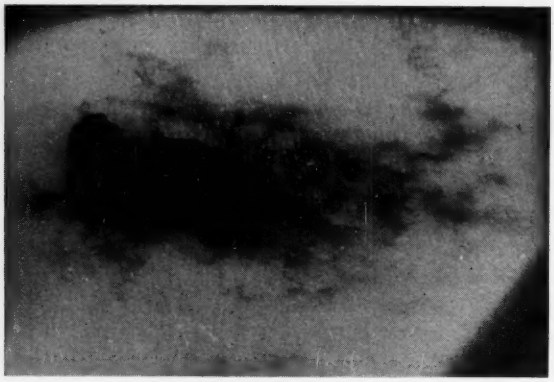

FIG. 133.—Same case, showing pattern immediately after injection into swollen forearm.

FIG. 134.—Same case, showing pattern 8 minutes after injection into swollen forearm.

FIG. 135.—Same case, showing general appearance at 20 minutes.

In the case of a patient operated on eight years previously, who had no swelling of the limb except occasionally on the inner aspect of the arm just above the elbow, the results may be taken as representative of the state of the lymphatics and the rate of lymph-flow in the affected arm of those patients whose arms do not swell, or do so to a slight extent. In these patients there is often evidence of slight dilatation of the cutaneous lymphatics on the affected side, and the rate

of streamer formation is slower but still nearly the same as that on the normal side. In addition, in this case it was noticed that the lax skin above the elbow, the site of occasional swelling, was picked out by a fine network of dye shortly after the injection in the forearm. This may be taken as evidence that in those areas of the limb which do swell, the lymphatics are dilated. It is tempting to attribute this to permanent occlusion of the main lymph-trunks draining that particular area. The details of this experiment are illustrated in *Figs.* 136, 137 (*Case* M. W.).

The appearance of an isolated part of a streamer at the elbow flexure crease, observed in some other cases also, is explained on the supposition that at this point the lymphatics are much nearer the skin, owing to the comparative absence of subcutaneous fat.

Observations on a third group of patients were made to determine the state of the lymphatics and the lymph-flow at varying periods after operation, and to study the influence of deep X-ray therapy and wound sepsis on the lymph-flow.

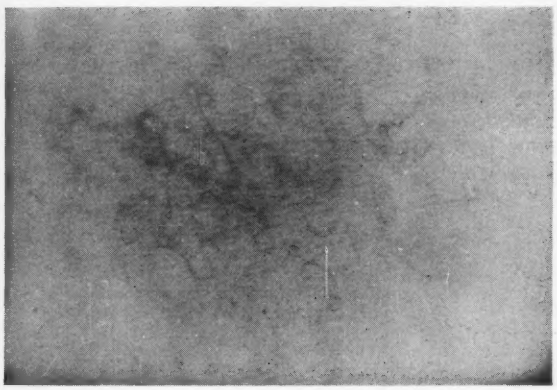

FIG. 136.—*Case* M. W. Showing pattern in affected limb, 16 cm. proximal to injection site, 5 minutes after injection.

FIG. 137.—Same case. General view at 10 minutes, showing isolated streamers at elbow crease and site of pattern shown in detail in *Fig.* 136 (indicated by arrows).

Of four patients studied after operation, i.e., at 5, 6, 16, and 19 days, the results were briefly as follows :—

In one the rate of lymph-flow in the affected limb was practically normal; in two it was diminished, but only moderately, and in one (at 19 days) it was reduced to zero. It is interesting to consider these results in the light of Reichert's findings (1926). Making a clean section across a hind limb of a dog, except for the femur, femoral artery, and femoral vein, he found that lymphatic regeneration was beginning on the fourth day and by the eight day was " physiologically adequate ". There was no wound infection in any of these three patients, and if their lymphatics had behaved like those of Reichert's dogs, we should have expected the patient H. B., tested 19 days after operation, to have shown the nearest approximation to the normal. We find, on the contrary, that in some patients, at any rate as early as 5 and 6 days after operation, the rate of lymph-flow

is nearly that of the normal, while in one, 19 days after operation, there is complete lymph-stasis which presumably dated from the time of operation. A possible explanation of these results will be discussed later.

Of the remaining three patients, one, R. D., had post-operative deep X-ray therapy; in this patient also the axillary end of the wound had suppurated. Healing by first intention had occurred in the other two. In both the latter cases there was evidence of slight impairment of the lymph-flow. In case A. W., *Figs.* 138–142 clearly show evidence of dilated and more easily filled lymphatics

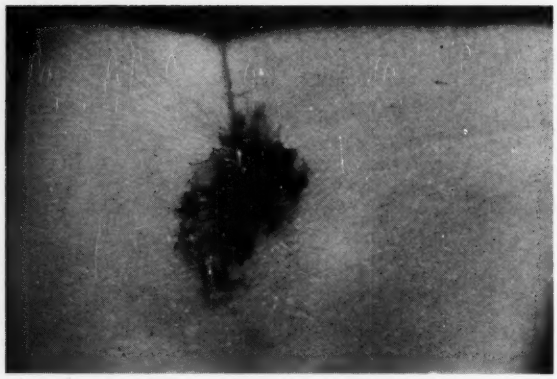

FIG. 138.—*Case* A. W. Pattern forming during injection of normal forearm.

FIG. 139.—Same case, showing injection pattern and streamer running to axilla 10 minutes after injection of normal arm.

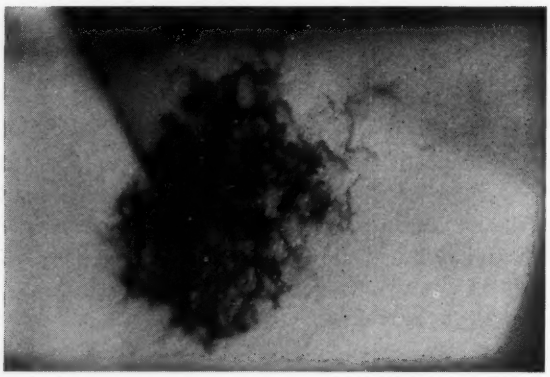

FIG. 140.—Same case. Pattern forming during injection of affected forearm, for comparison with *Fig.* 138.

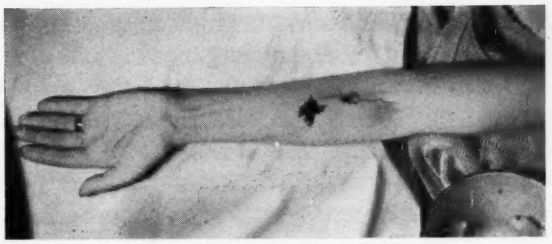

FIG. 141.—Same case. Streamer running to elbow 11 minutes after injection of affected forearm.

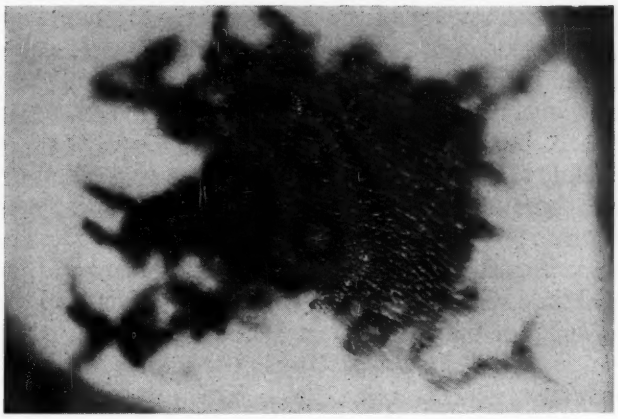

FIG. 142.—Same case. Injection pattern on affected forearm 10 minutes after injection, showing diffusion of dye.

on the affected side. In the case of the patient who had deep X-ray therapy and whose wound had suppurated, there was no evidence of interference with the lymph-flow. Thus, neither wound suppuration nor deep X-ray therapy necessarily interferes with the lymph-flow.

Before assuming that the absence of streamer formation and the demonstration of dilated lymphatics in the affected limbs of those patients with grossly swollen arms can be taken as evidence that the lymph stasis was the cause of the œdema, the possibility that the lymph stasis may accompany the œdema, and not cause it, must be considered. Thus, in the case of J. S., in which swelling of the arm followed an extensive thrombosis of the main venous channels draining the limb, there was evidence of complete lymph stasis accompanying the swelling and of the return of the lymph-flow when the swelling had subsided, and McMaster has shown that in cardiac œdema there is no active lymph-flow. As in the case of J. S., there was a thrombosis of the jugular and subclavian veins, there was presumably a complete block to the thoracic duct, and therefore at least some interference with the rate of lymph-flow from the left arm ; and again, as the thrombosis followed severe wound suppuration as the result of spontaneous œsophageal fistula, it is likely that the main afferent lymphatics were also thrombosed.

For these reasons we conclude that in this patient complete lymphatic obstruction was present in addition to venous. McMaster (1937) has shown that in cardiac œdema there is no active lymph-flow, and the lymphatics are dilated and more permeable than normal, yet in these cases it is clear that lymphatic obstruction is

not the cause of the œdema, and McMaster himself states that the " stagnation of lymph in the œdematous skin of the cardiac patient is not easily explained ". He suggests that it is due to valvular incompetence of the lymphatics, but concludes that complete proof of its existence is lacking. Now œdema alone will not bring about lymph stasis, for in nephritic œdema McMaster has found that the lymph-flow in the œdematous skin of nephritic patients is usually greater than normal. Veal (1938), citing no authority, and giving no evidence, states that " the œdema resulting from simple mechanical venous obstruction is similar to cardiac œdema both physically and chemically, and in the manner in which it is produced ", and goes on to assume that " in œdema of the arm resulting from venous obstruction the lymphatic circulation undergoes the same changes as occur in cardiac œdema ". But as Smirk (1935) has shown that in cardiac œdema two of the important factors are an increase in the capillary permeability and a fall in the colloid osmotic pressure of the plasma, and that the increases in general venous pressure in cases of congestive heart failure are not by themselves sufficiently great to cause œdema, and as Krogh et al. (1932) have shown that in venous congestion in normal subjects there was always a diminution in the rate of filtration as the colloid osmotic pressure of the blood increased, Veal's statement can hardly be accepted as correct.

Since our experiments show that the direction of change in the rate of lymph-flow in the affected limb of practically all the patients subjected to radical mastectomy was towards a slowing of the rate, and as in none of them was there any evidence of abnormality of the blood circulation through the limb, it would seem reasonable to conclude that the complete lymph stasis demonstrated in the grossly swollen arms was the cause of the œdema.

To sum up then, we may state that as a result of radical mastectomy there may be in the affected arm either no alteration, moderate diminution, or total cessation of the lymph-flow, and that in those cases in which total lymph stasis has occurred, this alone is sufficient to account for the persistent œdema, neither wound sepsis, deep X-ray therapy, nor recurrent attacks of inflammation being necessary for its development or persistence.

DISCUSSION

Œdema is a pathological condition in which fluid accumulates excessively in the tissues of the body (Loeb, 1923). Palpable œdema in a limb demonstrable by pitting on pressure occurs when the excess fluid increases the limb volume by 8 per cent (Drury and Jones, 1927).

It is commonly taught that there is some essential difference between so-called lymphatic œdema and œdema from other causes, and descriptions are given by which cases may be recognized and allotted to their appropriate categories. Thus Veal (1938) says " pure lymphatic œdema is of a brawny persistent type, which is only partially reduced by rest and elevation of the arm. The skin is firm and pits slightly on pressure, but does not wrinkle in the pit ", and " the œdema produced by venous obstruction is of the soft pitting type, the skin is thin and tense and there is wrinkling of the pitted portion."

Even if, as Drinker and Field (1933, *a*) predict, and as our observations confirm, in pure lymphatic obstruction the protein content of the œdema fluid is high (in two of our cases 2·99 and 2·17 g. per cent), that is no reason why the œdema should be of a different character as the protein is in solution. In

both cases referred to it is true that pitting was not easily obtained, but that this is not due to a high protein content of the œdema fluid is shown by the observation that in a patient with œdema of both legs of unknown aetiology, the protein content of the œdema fluid was 3·01 g. per cent, and in this case the œdema was soft and pitted easily on pressure. The characteristics 'hard', 'brawny', 'non-pitting' which are supposed to typify lymphatic œdema, may more simply be regarded as being due to a high tension of œdema fluid in the tissues, often found in cases with lymphatic œdema, since it is usually persistent, but which may occur in any long-standing œdema whatever the cause, as, for instance, in the legs of patients with chronic varicose ulcers. The reasons for arriving at this conclusion are threefold: if a case of 'lymphatic œdema' is watched from its inception, the œdema gradually changes from a soft easily pitting swelling to a hard brawny one which does not pit easily on pressure (*see Case* A. B., described above). If a patient with established 'lymphatic œdema' has the affected limb elevated for long enough, the reverse changes occur, and finally, œdema similar in all clinical aspects to 'lymphatic œdema' may accompany the chronic ulceration associated with idiopathic varicose veins. Such œdema will become soft and pitting and finally disappear after prolonged rest in bed, though, as in a case of this nature recently seen, the time spent in bed may have to be as long as three months. In this patient there was a residual thickening and induration of the subcutaneous tissue after the swelling had disappeared.

Though chronic œdema alone might produce slight fibrosis, it is probable, as Matas (1913) suggested, that lymphatic obstruction in a limb increases not only susceptibility to infection but the ease with which diffuse fibrosis of the subcutaneous tissue occurs following inflammation. Such a sequence of events seems to be necessary for the development of filarial elephantiasis (Grace and Grace, 1931), and occurs in some patients with swollen arms after radical mastectomy, but not in all. Halsted (1921) was of the opinion "that although blocking of the lymphatics and occasionally also of the veins was the underlying factor, infection played a conspicuous part in the determination of the amount of the swelling and the time of its manifestation". Yet his reasons for thinking so were not entirely convincing: "the infection may quite conceivably be so mild in degree as to escape the observation even of those constantly on the look out for it" (Halsted, 1921). While repeated attacks of inflammation in a swollen limb will tend still further to increase the swelling and fibrosis, yet it is clear that lymphatic obstruction alone will produce severely swollen limbs. The changes produced in the limb by attacks of inflammation should not be confused with those produced by lymphatic obstruction alone.

Yet before we can accept the view that lymphatic obstruction alone can produce a severely swollen arm, certain difficulties have to be faced. Why, for instance, should so few of the patients subjected to a nearly identical operation show any swelling; only 15 of 90 patients surviving for five years and over showed any swelling, and in only 7 was it moderate or severe (Devenish and Jessop, 1937). Again, how can one reconcile the facts of the rapidity of regeneration of lymphatics and the difficulty of producing lymphatic obstruction with the occasional production of lymphatic obstruction by radical mastectomy.

To take the last point first, Reichert showed that after complete division and suture of all the tissues of a dog's limb except the femur and the femoral artery and vein, new lymphatics could be shown to cross the scar by the fourth day, and by the eighth day "the regeneration is physiologically adequate. Where there

is any appreciable scar or where healing has been interfered with by trauma, foreign body, or infection, the growth of lymphatics across that region may be delayed for weeks or months". He was unable to produce any permanent œdema of the limb.

Pullinger and Florey (1937) have shown that while in the scar tissue itself produced by an injection of silica there is permanent obliteration of the lymphatic channels, yet in the immediate neighbourhood numerous fresh lymphatics appear and remain patent up to many months later. We should therefore expect that following the clearance of the axilla during radical mastectomy new anastomotic channels would be provided, and this no doubt happens in the majority of cases—in fact, infection of the axillary end of the wound should stimulate the formation of anastomotic lymph channels in the surrounding tissues. Reichert's experiments seem to provide the greater difficulty, yet it should be pointed out that in his experiments he made a clean circular cut across the limb and re-sutured it, while in radical mastectomy not only is the whole mass of regional lymphatic glands removed, but also varying lengths of the main lymphatics of the arm. That these two procedures are comparable is very doubtful. It is more difficult to reconcile Pullinger and Florey's observations with the facts we have observed, but we suggest the following explanation: The lymphatics in the subcutaneous tissue "are chiefly large draining trunks, with very few of the smaller vessels and very few capillary lymphatics. . . . The relation of these lymphatics to the deep fascia needs emphasis. They lie in the superficial fascia or fat, not directly on and rarely in the deep fascia" (Gray, 1939). These large draining trunks enter the axillary lymph-glands by perforating the deep fascia of the axilla over a relatively wide area. Now, during the operation for radical mastectomy a small variation in technique which is conditioned to some extent by the build of the patient, namely, variations in the amount of subcutaneous fat left on the axillary flap, may produce very considerable differences in the lengths of main-draining lymphatics removed. In thin patients a relatively thicker layer of subcutaneous fat will be left on the flaps, and consequently in these patients the large lymphatics in the subcutaneous tissue will be relatively undisturbed, while in fat patients there will be a much greater chance of removal of considerable lengths of main lymphatic trunks. When too many of the main lymphatics have been removed over too large an area we suggest that their regeneration may be inadequate to restore the lymph-flow. Consequently, we should expect severe swelling of the arm to be commoner in fat patients. This is, in fact, the case.

Late Onset.—Accepting the view that swelling of the upper limb following radical mastectomy is caused by extensive interruption of the lymphatic drainage at the time of operation, the frequent delay in the onset of the swelling requires an explanation. There was some delay in 5 out of 15 cases, varying from six weeks to sixteen years (Devenish and Jessop, 1937). It is possible that this is another aspect of the question why only one out of every six patients undergoing the same operation is affected.

Search was therefore made for a factor which could operate to delay the onset of the swelling. One such factor might be the elasticity of the skin of the limb. It was found that after reduction of the swelling comparatively light pressure by a corset was all that was necessary to control the swelling even when of a severe degree. It is possible that such pressure may be maintained in the majority of

cases by the skin itself, and that the swelling will only develop when the elasticity of the skin diminishes.

In some of the cases investigated by the dye test in which swelling had not occurred, it was found that lymph-flow was slowed but not abolished on the operated side. Now it is well known that the elasticity of the skin diminishes with increasing age. It would seem reasonable, therefore, to suggest that the late onset of the swelling may be due to a combination of such partial lymphatic obstruction and loss of skin elasticity.

Distribution.—In the severely swollen limbs constrictions were always noted at the elbow and the wrist. This appears to be due to two factors—namely, the tethering of the skin to the deep fascia at the flexure creases (Wood Jones, 1920), and the effect of constant movement of these joints in keeping the overlying tissues free from fluid.

In moderately swollen limbs the swelling is confined to certain areas, which might seem to indicate that in such areas the tissues were more easily destructible than elsewhere. An attempt was made to verify this suggestion experimentally by measuring the rate of flow of normal saline through a needle into the subcutaneous tissues in different parts of the limb. The results were not conclusive.

Variability in the Size of the Swelling.—Most patients with swollen limbs found that the swelling increased after work or at the end of the day or after walking (Devenish and Jessop, 1937). As Drinker and Field (1933, *b*) point out, "there can be no doubt that lymph production increases under circumstances of increased filtration from the blood capillaries such as is shown to accompany muscular work". Such excess fluid must accumulate in the tissues, with a resultant increase in the size of the limb unless the excess fluid is removed at a rate equal to that of its formation. Krogh et al. (1932) have shown that the rate at which fluid was removed from the tissue spaces depended on the size of the accumulation of fluid, being distinctly more rapid with large amounts. They found that small amounts were removed by true absorption at the venous end of the capillaries, while large amounts were removed by the lymphatics. Now in the patients we are considering, the accumulation of fluid in the swollen limb is large (*see Table II*). Hence the presence of lymphatic obstruction must result in increased swelling of the limb, when the tissue fluid is increased as the result of exercise. When the limb is at rest removal of some of the excess fluid must be carried out by true absorption at the venous end of the capillaries, and consequently the swelling will tend to be smaller at the end of such periods, namely, in the morning. Variations in the amount of the swelling with exercise may thus be attributed solely to the presence of lymphatic obstruction.

Finally, we may consider as a practical problem whether any modification of the operation of radical mastectomy which would diminish the liability to post-operative swelling of the limb is possible. It is clear that any such modification must not contravert the sound pathological principles upon which the present operation is based. Thus, whilst excision of all the lymphatics running from the breast to the axilla is essential, it might be possible deliberately to preserve some of the main lymphatic channels at the upper end of the arm. As we have previously pointed out, these main lymphatic channels run in the fatty subcutaneous tissue of the axillary flap. These are, therefore, more likely to be preserved if the subcutaneous tissue is left in the flaps and their reflection carried out by dissection in a plane immediately superficial to the deep fascia. This should only be done

on the upper flap, and from the axillary end of the incision to a point midway along the clavicle. Such a modification, whilst not interfering with the radical extirpation of the lymphatics draining the breast, should diminish the incidence of this distressing complication. This modification is particularly desirable in the case of fat patients in whom severe swelling is more prone to develop.

SUMMARY

1. Post-operative non-malignant swelling of the upper limb is due almost entirely to an accumulation of fluid in the subcutaneous tissues. The possibility of a slight increase in subcutaneous fibrous tissue has not been excluded.

2. There was no evidence of venous obstruction in the cases investigated. Increased venous pressure occurring during exercise is a contributing factor to the swelling only in the presence of lymphatic obstruction.

3. Post-operative lymph-flow was investigated by dye injections of the skin lymphatics. It may be unaltered, diminished, or stopped.

4. Lymphatic obstruction alone is sufficient to cause the swelling. Neither wound sepsis, nor recurrent attacks of inflammation in the limb, nor deep X-ray therapy are necessary for its development or persistence.

5. The characteristics 'hard', 'brawny', 'non-pitting' supposed to typify lymphatic œdema may be entirely due to a high degree of tension of œdema fluid in the tissues.

6. The infrequent development of the swelling following a standardized operation may be due to variations in the extent to which the main lymphatic trunks draining the upper limb are excised.

7. Delayed onset may be due to a combination of partial lymphatic obstruction and loss of skin elasticity.

8. Preservation of the main lymphatic trunks by leaving a sufficient thickness of subcutaneous tissue on the upper axillary flap should diminish the incidence of the complications.

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VOLKMANN'S ISCHAEMIC CONTRACTURE*

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Until the last quarter of the past century any muscle contracture occurring after an injury to an arm or a leg appears to have been regarded as invariably due to nerve paralysis. Occasional cases had been reported by such surgeons as von Stromeyer (1840), Guérin (1842), and others in which the contracture had appeared unusually early after the accident, but not until 1872 was there any suggestion that these contractures could be due to any other cause. In that year Richard von Volkmann (1872) (*Fig.* 143) published the first specific account of a rapid post-traumatic muscle contracture of acute onset, reaching its climax in a few weeks, and progressing relentlessly despite the sort of passive movements and splinting which would prevent the slower, later, and less severe contracture resulting from nerve injury. In this and in his second paper (1875), Volkmann regarded his syndrome as due to an "inflammatory myositis", but in 1881, in his third and most definite paper, he stated categorically that these rapid contractures, though always associated with paralysis, "must be regarded as ischæmic". He held that they were caused "by a continuous stoppage of the arterial blood", for which he blamed tight splinting, and he showed the change in the muscle to be necrotic and not inflammatory.

Fig. 143.—Richard von Volkmann.

In 1884 Leser described 7 cases occurring in Volkmann's clinic and gave a clear account of the clinical features. The term 'Volkmann's contracture' was first used by Hildebrand in 1890. It came into use in England after papers by Wallis (1901) and Dudgeon (1902). By 1909 J. J. Thomas had been able to collect 107 case reports from the literature, and the condition could be termed "well recognized".

Incidence.—The extreme rarity of Volkmann's ischæmic contracture in an organized fracture clinic in this country is illustrated in *Table I*, which shows that only 8 of 21,000 fractures presenting at Professor Platt's clinics for their primary treatment developed this condition. No case at all has occurred in the last eight

* Being a Hunterian Lecture to the Royal College of Surgeons of England, February 2, 1940.

Table I.—INCIDENCE OF VOLKMANN'S ISCHÆMIC CONTRACTURE
(*Fracture Services of Professor H. Platt, Ancoats Hospital, Manchester*, 1922–33; *Manchester Royal Infirmary*, 1934–38)

Total number of recent fractures receiving primary treatment in these clinics		21,000
Of which, fractures of the lower end of the humerus were	1655	
And of both bones of the forearm were	2127	
Number of these cases developing Volkmann's contracture (4 very mild, 2 mild, 2 severe)		8

thousand fractures treated in this service. Three additional cases have presented at these clinics from twenty-four hours to four days after the receipt of fractures which had been manipulated and immobilized elsewhere, and 18 cases have arrived more than three and a half weeks after their injuries with contractures which were already well established. Bittner (*vide* M. Kappis, 1938) reports a similar rarity, 4 ischæmic contractures in 2850 cases of fracture of the upper limb, but in some clinics the condition appears to be far commoner. Putti (1938) has had 16 cases in 55 consecutive elbow fractures.

Antecedent Injuries.—While the condition almost always follows a fracture, Volkmann himself (1895) reported instances which had occurred following injury to the soft parts alone. In 1901 Wallis reported a case after a prolonged operation under an Esmarch bandage. R. Jones (1907), Bérard, Policard, and Wertheimer (1924), and many others have also seen cases in which no fracture at all had occurred, and I attach considerable importance to three patients, discussed later, who acquired the condition after arterial embolism.

Among fractures, the extension fracture of the lower end of the humerus ('supracondylar' fracture) has provided the majority of the reported cases, while fractures of the shafts of one or both forearm bones have been responsible for about

Table II.—ANTECEDENT LESIONS IN 32 MANCHESTER CASES OF VOLKMANN'S ISCHÆMIC CONTRACTURE

CASES FOLLOWING INJURY:—	*Number of Cases*
a. Elbow—	
Supracondylar fracture of the humerus	12
Dislocation of the elbow	1
Fracture of the olecranon	1
Fracture of the upper third of the ulna with dislocation of the head of the radius	1
Total after elbow injuries	15
b. Forearm—	
Fracture of the shafts of the radius and ulna	11
Fracture of the shaft of the radius alone	1
c. Wrist—	
Fracture of the lower end of the shaft of the radius	1
d. Whole upper limb—	
Crush injury of the upper arm and forearm	1
Total cases following injury	29
CASES FOLLOWING ARTERIAL EMBOLISM:—	
a. Brachial artery	1
b. Common femoral artery	2
Total cases following embolism	3

30 per cent. The condition has, however, followed fractures of the clavicle, humeral shaft, olecranon, wrist-joint, and leg bones (Thomas, 1909, Rocher, 1937, Putti, 1938, et al.). In the present series of 32 cases, the antecedent lesions are stated in the table (*Table II*), which shows a slightly higher incidence after forearm fractures than might be expected.

Age Incidence.—The age incidence in this series is illustrated in *Table III*, and presents no unusual features. The lesion is commonest between the ages of 2 and 16, and one certain reason for its great rarity in adults lies in the fact that forearm fractures and the extension supracondylar fracture are both very much commoner in children. The oldest recorded case, a man aged 50, is noted in Thomas's (1909) paper.

Table III.—Age Incidence in 29 Post-Traumatic Cases of Volkmann's Ischæmic Contracture

First decade	18 cases
Second decade	9 cases
Third decade	1 case
Fourth decade	1 case

Youngest cases (2) were 3 years of age.
Oldest case was 38 years of age.

CLINICAL FEATURES

The clinical features of Volkmann's contracture are so well known that detailed description would be superfluous. Certain facts, however, must be stressed.

While the first symptom or sign usually appears within a few hours (two and a half hours was our earliest) of the injury or of the reduction of the fracture, the onset may be delayed as long as twenty-four hours or even a few days. Not every case starts with the burning pain in the hand or forearm which is usually described. Especially where there is a major nerve injury, the onset may be quite painless, and even if pain is severe there may be considerable and deceptive relief after forty-eight hours or so.

Pain in the forearm on passive extension of the fingers is a very serious early sign, as is the assumption of a flexed position in these digits. It is my belief that either of these signs indicates that severe damage has already been done to the flexor muscles. If preventive treatment is to be attempted with any hope, earlier signs must be sought.

The most important of these are the signs of obstruction to the circulation of the limb, usually in the form of cyanosis and swelling of the fingers. Blue and swollen fingers are, however, by no means inevitable. I have had the mortification of watching a severe ischæmic contracture develop in a boy whom I had treated from the start, whose hand showed no cyanosis of any note until the third day, while P. D. Wilson (1933) and Jones and Lovett (1929) both mention *pallor* of the hand as a not infrequent early sign, which I regard as of great importance.

It is my belief that the radial pulse is probably constantly absent in all severe cases in the first three or four days. Since 1926 there have only been 4 cases of Volkmann's contracture in Professor Platt's clinics within four days of the initial injury, and though the state of the radial pulse was not recorded in two of these (one of which was seen thirteen years ago), in the other two it was demonstrably

absent. One of these cases recovered his radial pulse on the fifth day*, so that the presence of a palpable radial pulse in a later case is of limited significance. Even so, three of the six chronic cases I have seen in the last three years have had no palpable pulse. Case reports in the literature unfortunately rarely mention the radial pulse, but since 1931, in eleven papers,† 27 early cases have been reported in which the radial pulse was absent, while in 1 only (Tavernier et al., 1936) has it been reported to be present, and even then it was weak. In the chronic stage persistence of the absent pulse had been reported by Petersen (1888), Thomas (1909) in 8 out of 10 cases, Leriche (1928), Meyerding (1936), Guilleminet (1939), and by several other observers.

If the radial pulse itself should be present, examination with the oscillometer of Pachon (*Fig.* 144) may be expected to show evidence of impaired arterial circulation at all stages (Massart, 1935). While I have not had the opportunity to use

FIG. 144.—The oscillometer of Pachon. The small dial records the pressure in the armlet; the large dial records the oscillations of the pulse wave when the pressure in the armlet is intermediate between the systolic and diastolic arterial blood-pressure.

this instrument in acute cases, it shows a constant impairment of oscillations in the affected limb in all chronic cases. The instrument consists of a double aneroid for demonstrating the oscillations of the pulse-wave in a limb. Typical findings are shown in *Fig.* 145, which exhibit depressed oscillations even in chronic cases in which the radial pulses were clearly palpable.

A skin thermometer will demonstrate a constant depression in the surface temperature of the affected limb. Indeed these limbs sometimes behave as if they were cold-blooded.

The other features of the chronic, established contracture require no comment. A typical established deformity is illustrated in *Fig.* 165.

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* The radial pulse has also returned by the fifth day in three personal cases of ligature of the brachial artery at the elbow for hæmorrhage.

† Aurousseau (1938), Fèvre and Bertrand (1935), Fleming (1931), Fontaine and Kunlin (1936), Funck-Brentano (1935), Gabrielle (1938), S. Jones (1935), Leveuf (1938), Pouzet and Leclerc (1937), Tavernier (1936), P. D. Wilson (1933).

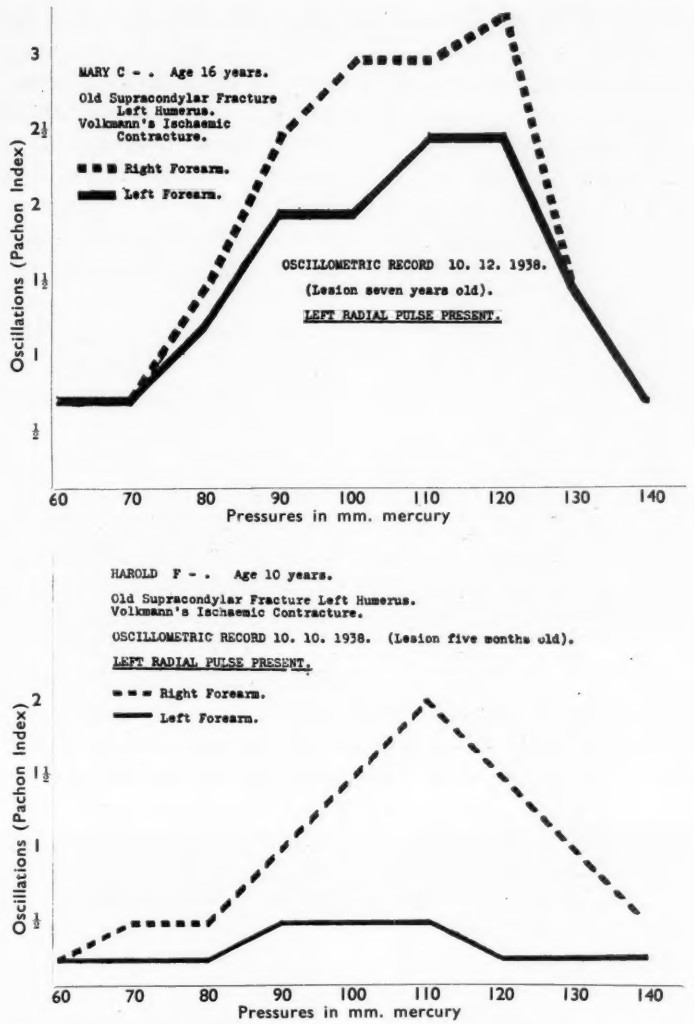


FIG. 145.—Oscillometric records from two cases of established ischæmic contracture following supracondylar fractures. Both cases showed easily palpable radial pulses.

MORBID ANATOMY

The naked-eye and microscopical appearances of the affected muscle in established cases of Volkmann's contracture are both quite typical. These changes were fully described by Greenfield in a case reported by Bristow (1923), and the present series confirms Greenfield's findings. Occasionally, as in a case of seven years' history with a remarkable recovery of muscle power, there may be a fair amount of recognizable skeletal muscle present in the forearm, but usually the flexor mass does not look like muscle at all. Yellow-green in colour, it is surrounded by a dense fibrous tissue sheath, inside which one finds the centre of the muscle belly replaced by a hard homogeneous yellowish substance, which cuts with difficulty (*Fig.* 146). This change is established early. I have seen it in a case only four days old, following a fracture of the forearm bones, already well established,

though strikingly localized to the area distal to the fracture. It is found without fail in all established cases.

The histology is equally constant. In early cases there may be small areas of white-cell and round-cell infiltration, and various degrees of dilatation of venules and arterioles, but, though a great deal of importance is attached to these changes by Leveuf (1937, 1938) and by Tavernier (1938), I regard them as of doubtful significance. What is really important in these early cases is a considerable reduction in the nuclei of the muscle-fibres over certain areas, the 'islands of necrosis'. By the time the chronic stage is reached this loss of nuclei has become complete. One sees masses of necrosed muscle in which individual fibres are recognizable, but there is hardly a live nucleus even in the inter-fibrillar fibrous tissue (*Fig.* 147). These masses resemble nothing more closely than a muscle sequestrum, upon the surface of which is a sheath of dense fibrous tissue. Between this sheath and the dead fibres themselves there is an area of cellular activity, both fibroblastic and phagocytic (*Figs.* 148, 149). The latter function is performed by macrophages and large muscle giant-cells. It is absolutely characteristic of this lesion that all cellular activity and all fibrosis occur at the periphery of the deadmuscle masses.

FIG. 146.—Transverse section of a whole pronator teres in an ischæmic contracture of eight months' history, showing the central massive necrosis and the surrounding fibrous tissue.

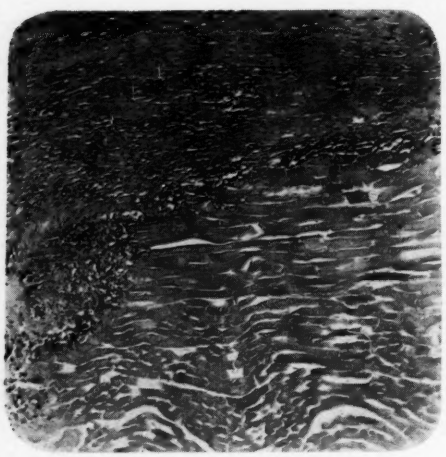


FIG. 147.—Low-power view of the flexor sublimis digitorum in a case of ischæmic contracture explored eight months after a supracondylar fracture of the right humerus. Note the completely necrotic muscle in the right lower quadrant of the field, with dense scar tissue on the periphery of the muscle and cellular activity at the junction of muscle and fibrous tissue. (× 36.)

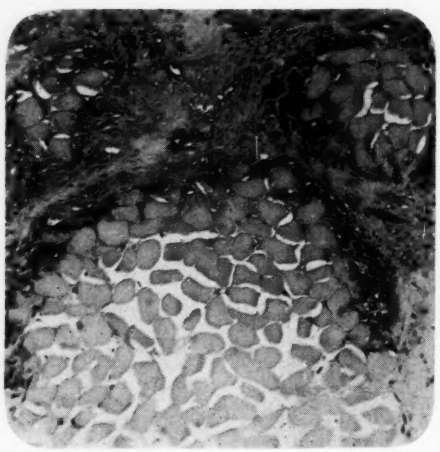


FIG. 148.—Transverse section of the flexor sublimis digitorum in a case of five months' history, showing necrosis and fibrosis around muscle bundles (*see also Fig.* 163).

The process differs completely from all other muscle changes except one—infarction. In muscle atrophy or contracture after denervation (*Fig.* 150) or sepsis there is interfibrillary fibrosis, which is *never* seen in Volkmann's contracture, and these conditions never produce the typical massive muscle sequestrum of the latter lesion.

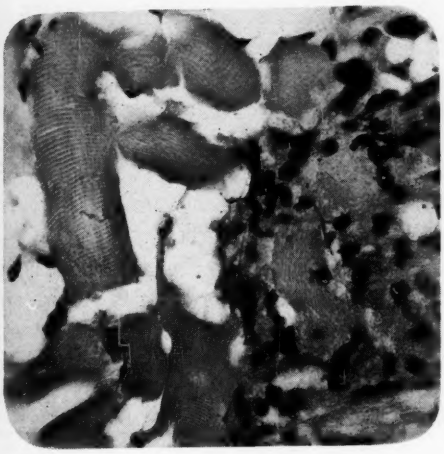


FIG. 149.—High-power view from the same section as *Fig.* 147, showing phagocytic activity at the edge of the muscle 'sequestrum'. Note the preservation of striations, which are seen only at the extreme edges of the necrotic masses. (× 400.)

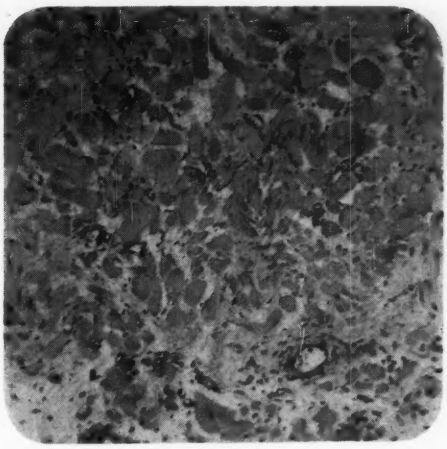


FIG. 150.—Histology of flexor carpi ulnaris after long-standing division of the ulnar nerve. Note deposition of young fibrous tissue between the individual fibres, together with atrophy and interfibrillar cellular activity. These features are completely different from those of ischæmic contracture. Contrast with *Figs.* 147, 148.

Eventually fibrosis proceeds from without inwards and completely engulfs the muscle bundle. Any regeneration or repair which may occur appears to be solely the result of hypertrophy of muscle which was presumably undamaged in the original lesion.

DIAGNOSIS

In a typical and established case clinical diagnosis is usually absurdly easy. Difficulty can only arise from the claw hand of a median and ulnar palsy or from the dropped wrist or fingers of a musculospiral or posterior interosseous nerve lesion, particularly if these lesions should coexist with the ischæmic contracture. Contractures of the flexor muscles due to scarring after infected wounds may resemble Volkmann's contracture, and have, indeed, been reported as such, I believe erroneously—Hamada (1937), de Araujo (1937). Hæmophiliac hæmorrhages in the flexor muscles occurring in the absence of severe trauma have led to contractures which have been reported as Volkmann's—Hey Groves (1907), Frölich (1925), Pasquali (1931), Hsi-en-Shih (1931), and Hill and Brooks (1936) —but in all these cases the clinical history has failed to impress me as conclusive, and histological confirmation is, of course, lacking.

In really early cases accurate diagnosis is a matter of urgent importance. The signs I have already described must be the surgeon's guide, especially the absence of radial pulsation and the weakening of oscillations.

PATHOGENESIS

External Compression.—Volkmann's original theory that the contracture is usually due to excessively tight bandages or splints is easily disturbed. As early as 1888 Petersen reported a case in which no constricting agent had been applied to the arm at any time. Since then Robert Jones (1907), Miltner (1931), Hill and Brooks (1936), and many others have reported over a hundred cases in which there has been no external pressure at all. Indeed, many of these cases have been treated in nothing more than a sling. It must, however, be admitted that so many cases *have* followed tight splinting that only a bold man would acquit this factor completely. Leveuf (1938) reserves the name "true Volkmann syndrome" for the cases which have been tightly splinted, terming the others "soi-disant syndrome", but histologically both these are the same lesion. I can only regard external constriction as, at most, a contributory factor. Garber (1937) studied 80 consecutive cases of fracture of both bones of the forearm immobilized in plaster-of-Paris in Professor Platt's clinics, all of which came under my observation and most of which I had reduced. In 28 of these there was clear evidence of some undue tightness of the plaster, yet not one of them developed Volkmann's contracture. The only case which did, showed no signs of local or general plaster constriction, and there was a history of tight splinting in no more than eight of the present series of contractures.

Nerve Injury.—There are those who still hold that Volkmann's syndrome is, after all, merely an unusual variety of contracture due to nerve injury. It is undoubtedly true that very many cases, particularly the severe ones, have nerve injuries in addition to the contracture. In Thomas's (1909) review such nerve lesions were reported in sixty per cent of 107 cases, and Putti (1938) has actually had to operate on 44 nerve lesions in 58 cases. *Table IV* shows that 14 of my 29

Table IV.—Incidence of Nerve Lesions in 29 Post-traumatic Cases of Volkmann's Ischæmic Contracture

	Degree of Contracture			
	Severe	Moderate	Mild	Very Mild
Total number of cases	13	8	2	6
Nerve lesions :—				
Complete median and complete ulnar	3	1	–	–
Complete median and partial ulnar	–	1	–	–
Complete median alone	2	–	–	–
Complete musculospiral and partial median	1	–	–	–
Partial median and partial ulnar	1	–	–	–
Partial median alone	3	1	–	–
Partial ulnar alone	–	1	–	–
Total nerve lesions, single and combined	10	4	0	0

cases, and 10 of the 13 severe ones, had demonstrable lesions of the peripheral nerves of the forearm. So high an incidence of nerve injuries has led Putti (1938) to declare that the condition "is dominated by the lesions of the peripheral nerves", and that "the vascular factor . . . is of secondary importance" ("è dominata dalle lesioni dei nervi perifici . . . Il fattore vascolare . . . ha importanza secondaria"), a theory which is immediately upset by the many reported cases which have no nerve lesion at all. An admirable paper by Tower (1939) gives a full description of the contracture following simple denervation of muscle, and

absolutely reinforces Volkmann's original clear differentiation between the two conditions. In addition, it must be stressed that the histology of denervation shows atrophy and interfibrillar fibrosis, while that of Volkmann's contracture shows mass necrosis and fibrosis around necrosed masses.

Venous Obstruction.—In 1914 J. B. Murphy published the view that venous occlusion was the direct cause of Volkmann's contracture and that arterial obstruction by splint pressure plays " little or no role ". This view has received almost universal acceptance, particularly after the experimental work of Brooks (1922), Jepson (1926), and Middleton (1930). Brooks ligatured the entire venous drainage from the isolated rectus femoris of the dog, and the muscle duly developed a contracture which was regarded as being identical with Volkmann's. Ligature of the arterial supply of the similarly isolated muscle merely produced atrophy. Middleton (1930) repeated these experiments on thirty dogs and entirely supported Brooks's views, but, quite apart from the evidence I shall bring to show that these experiments bear no resemblance to the condition found at operation on human subjects, I cannot accept the conclusion that the lesion produced was a Volkmann's contracture. The histology of Middleton's contracture indicates at once that what was produced was an atrophy with an interfibrillar fibrosis, absolutely different from Volkmann's contracture (*Fig.* 151).

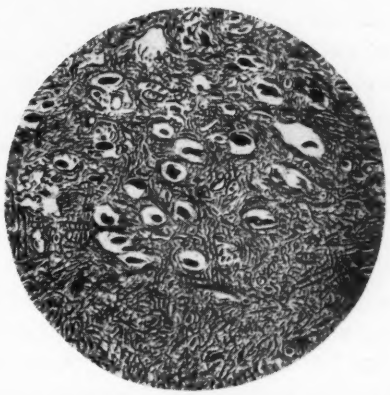


FIG. 151.—Histology of dog sartorius eight weeks after the Brooks-Middleton experiment. Note the interfibrillar fibrosis, quite unlike the histology of ischæmic contracture. Contrast with *Figs.* 147, 148, and 162. (*Reproduced by kind permission of Mr. D. S. Middleton and 'The British Journal of Surgery'.*)

Jepson (1926) ligatured the dog's femoral vein, made an incision one-third encircling the thigh down to the deep fascia, and applied an Esmarch bandage for twenty-four hours eight or ten days later. This experiment is far remote from clinical conditions. Only one dog appears to have shown a contracture which was at all lasting, and Jepson's photomicrographs are most unlike the histology of Volkmann's contracture, but this work has again and again been regarded as conclusive evidence that venous occlusion causes the Volkmann syndrome.

Alone of the workers on the venous side, Wertheimer, Déchaume, and Frieh (1937) have succeeded in producing a contracture with a histology like Volkmann's. Their experiment, blocking the terminal venules with a suspension of charcoal in gelatine injected into a vein under pressure, was, however, tantamount to capillary obstruction, and they themselves make no claim to be supporting a 'venous occlusion' theory.

Venous Occlusion as a Clinical Entity.—

a. The subfascial hæmatoma.—The findings at operation on acute cases of the contracture are completely at variance with the theory of venous blockage. Middleton (1928) described the venous drainage of the human forearm flexors as converging upon one vein, the deep median vein. This, he held, can become compressed by a tight hæmatoma under the fascial roof of the antecubital fossa in a supracondylar fracture, which thus occludes the venous return from the muscles

and produces Volkmann's contracture. This theory simply cannot stand. In all the reports of early cases operated upon in this country and elsewhere, the discovery of such a hæmatoma is a very rare event. Instead, what has been found by operators exploring early cases of the Volkmann syndrome is arterial injury and arterial obstruction. Extravasations were of course found in the substance of the muscles, as after any fracture, but even these are much more common in the brachialis anticus than in the forearm flexors. Moreover, it must be stressed that a hæmatoma *in a muscle* is a very different thing from a tight hæmatoma under the roof of the antecubital fossa. If a hæmatoma in a muscle causes Volkmann's contracture, then " all our athletes should be crippled " (Meehan, 1936).

b. The lesions found in acute cases.—Since 1930 28 cases have been reported in thirteen papers in which impending Volkmann's contractures after supracondylar humeral fractures were explored within the first ten days. Some of these operations took place within the first twenty-four hours. In 24 cases no subfascial hæmatoma at all was found. There were, as usual, subcutaneous extravasations and hæmatomata around the fractures, but even these were neither large nor tense, nor were they occluding any main vein. Every one of these cases, however, had impaired or absent radial pulses, and in 20 lesions of the brachial artery were found, varying from obstruction by spasm, incarceration of the vessel between the fragments, or angulation of it over the lower end of the upper fragment, to laceration or complete division at the site of fracture.* Only 4† early cases are reported in which any subfascial hæmatoma was found, and in 3 of these 4 cases major laceration and obstruction of the brachial artery was present. In the fourth case, in which the state of the artery is not reported (S. G. Jones's case), there was no radial pulse. A normal brachial artery in the first few days of a Volkmann's contracture has been found only by one man (Tavernier, 1938). My own experience of early operations upon developing Volkmann's contracture is limited to the four-day case which I have mentioned. This operation took place upon the forearm of a boy of nine with a fracture of the radial and ulnar shafts. Removal of the plaster, which was nowhere tight, enabled us to recognize absence of the radial pulse, and wide exploration of the forearm showed no evidence of intrinsic venous obstruction or of localized hæmatoma. What we did find was a tight spasm and total pulselessness of the radial, ulnar, and lower end of the brachial arteries, the radial artery being stretched over the lower radial fragment. Unfortunately, we were quite unable to relieve this arterial spasm, and the boy developed a severe Volkmann claw.

Three years earlier an impending Volkmann syndrome in a man of thirty had been explored by one of my colleagues. The syndrome followed a ' Monteggia ' fracture and was explored twenty-three hours after the receipt of this. Again, no hæmatoma and no trace of venous occlusion was found, but there was a significant feebleness in the pulsation of the brachial artery. A mild degree of contracture ensued.

c. The lesions found in chronic cases.—The discovery of an apparently normal artery in an old-established case of the contracture does not prove that the vessel

* Aurousseau (1937), Fèvre and Bertrand (1935), Fleming (1931), Fontaine and Branzen (1937), Funck-Brentano (1938), Girdlestone (1932), S. G. Jones (1935), Leveuf (1937, 1938), Pouzet and Leclerc (1937).

† Fontaine and Kunlin (1936), Gabrielle (1938), S. G. Jones (1939), H. P. Nelson (1930).

was not contused, perhaps severely, at the time of the fracture. Even so, however, a normal artery is rarely found even in chronic cases. Ever since Petersen's case in 1888, very many severely damaged arteries have been found at operation months or years after the original injury. Typical cases are reported by Guilleminet (1939),

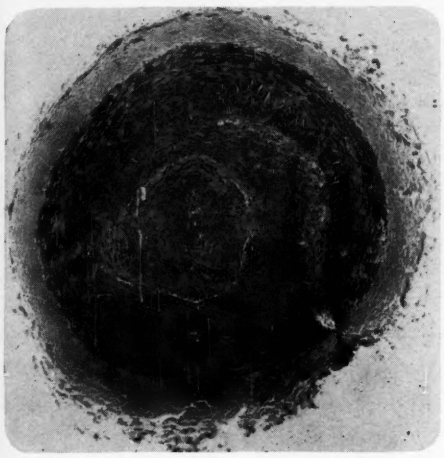


FIG. 152.—Thrombosis of the brachial artery in a case of established ischæmic contracture Same case as *Fig.* 147. (× 24.)

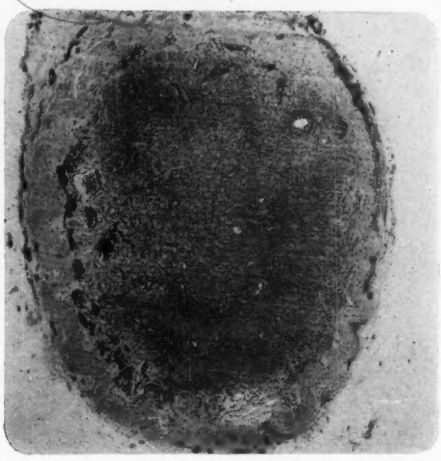


FIG. 153.—A high degree of thrombosis of the brachial artery in an established ischæmic contracture following supracondylar fracture of the humerus. (× 20.)

Leriche (1928), Mathieu (1934), Picot (1933), and many others, who have found old ruptures, lacerations, and thromboses of the brachial artery. In the last three years we have explored the vessels of 4 of the chronic cases in the present series eight months to seven years after supracondylar fractures. In 3 of these the original arterial injury had been so severe as to leave permanent evidence of damage, thrombosis of high degree in 2 cases (*Figs.* 152, 153), and rupture of the internal elastic lamina in 1 case (*Fig.* 154). In this third case and in the fourth the artery was buried in dense scar tissue, and though the fourth artery was of normal calibre and pulsating well, we have no histological evidence to prove its complete integrity.

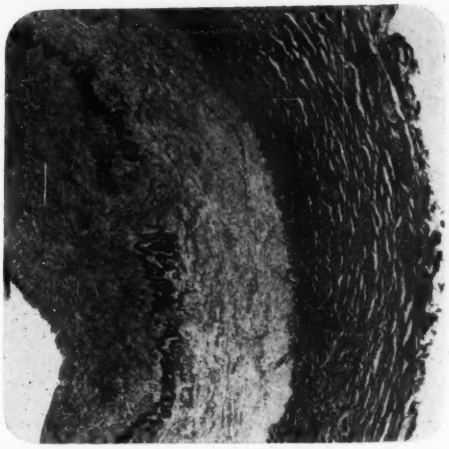


FIG. 154.—Histology of the brachial artery in a case of five months' history, showing an old traumatic rupture of the internal elastic lamina. The radial pulse was palpable. Same case as *Figs.* 148 and 163.

Volkmann's Contracture after Arterial Injury and Embolism.—In embolism of the major limb arteries Nature provides us with a unique human experiment illustrating the effects of pure arterial occlusion, and it has been my good fortune to be able to study an unusual number of such embolisms, for since 1925 Professor Jefferson and I have operated, together or separately, upon 20 cases, with results shown in *Table V*. Three patients, despite apparently successful restoration of the circulation, developed typical Volkmann's contracture after embolectomy.

Table V.—Operations for Arterial Embolism, 1925–40
(*Jefferson and Griffiths*)

Number of cases seen within forty-eight hours of onset of symptoms of embolism		21
Number of embolisms in these patients		27
Number of operations undertaken (in 16 patients)		20
Results :—		
a. Complete failure (post-operative gangrene or death)		8
b. Incomplete success (limited post-operative gangrene)		1
c. Complete restoration of circulation—		
After upper limb embolism	4	
After lower limb embolism	4	11
After aortic embolism	3	

Two of the above successes and one case which became a failure—three in all—*developed contractures*, one of the hand and forearm, two of the calf and foot.

The first of these occurred (Jefferson, 1934) in 1932, in a man of forty-two, with an embolism of the left arm due to auricular fibrillation. Professor Jefferson removed a clot from the bifurcation of the brachial artery two and a half hours after it had lodged there. The thrombus extended down into the upper ends of the radial and ulnar arteries so as to produce a high degree of obstruction to the collateral circulation. The operation restored the circulation and the radial pulse, but the patient rapidly developed a contracture of moderate severity in the forearm flexors, with a typical Volkmann claw and firm resistance to correction. The syndrome commenced within forty-eight hours of the embolectomy, and at no stage and at no level had the man any venous obstruction at all.

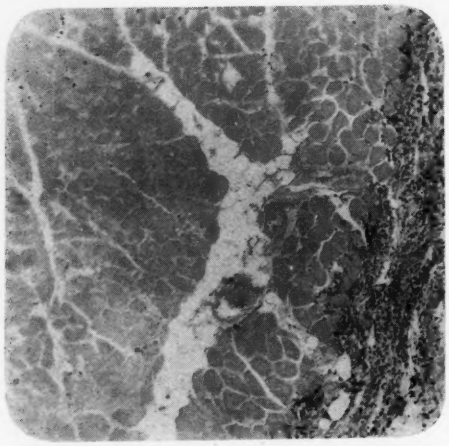


Fig. 155.—Histology of the soleus in a case of ischæmic contracture of the calf following common femoral embolism. The contracture was four weeks old. Note massive necrosis of the muscle with cellular activity at the periphery.

In 1937 Jefferson and I (Griffiths, 1938) removed an embolus which had been lodged for seventeen hours in the common femoral artery of a man of thirty-seven. Again, gangrene was completely averted and the circulation restored, though the posterior tibial pulse never returned. Forty-eight hours after operation the ankle and foot were acutely plantar flexed, the toes were cocked-up and flexed in a severe claw, and the calf muscles were hard, painful, and tender. Passive dorsiflexion was quite impossible because of the resistant contracture of the calf flexors. It took eighteen months to cure the contracture, even with the aid of wrenching and plastering under anæsthesia.

In the third case, in June, 1939, I removed a left common femoral embolus eight hours after it had lodged, in a woman of thirty-two who had had at least three previous embolisms in that limb. I found the profunda femoris thrombosed. Again, a typical Volkmann syndrome, exactly like the last, commenced within thirty hours, but it was cut short in this case by another embolism on the fifth day which led to gangrene of the foot. This conclusion, though unhappy, enabled

me to secure material for the histology of the flexor muscles, which is absolutely typical of Volkmann's contracture (*Fig.* 155).

These three cases should be considered with a similar report of Volkmann's contracture after embolism by Langer as early as 1895, and with such reports as there are of contracture after gunshot wounds of the main limb arteries.* In the light of these other cases and of the clinical and operative evidence, they bring conclusive proof that Volkmann's contracture is due to arterial occlusion and to nothing else.

Mechanism of the Ischæmia.—Arterial injury has been a well-recognized complication of fracture for many years. Usually it appears to have no permanent ill effect upon the limb; rarely it leads to gangrene. Exactly similar arterial lesions to those found in Volkmann's contracture have been found by Guilleminet (1937), Leveuf (1938), and Morwood (1939) in association with supracondylar fractures, but with no sign, symptom, or suggestion of Volkmann's contracture or of gangrene. Dodd (1934), on the other hand, reported 35 cases of gangrene following similar arterial injuries in various fractures, and Pampari (1936) and I have each once seen gangrene result from laceration of the brachial artery in supracondylar fractures in children, lesions identical with the ones I have described in Volkmann's contracture. How, then, does arterial injury sometimes produce no gross circulatory embarrassment, sometimes gangrene, and sometimes Volkmann's contracture, just as embolism can have any one of these three effects? The answer must lie in the collateral circulation. If this be free, all is well. If it be blocked completely, gangrene results. If the blockage be incomplete, but too severe or too prolonged for complete recovery of the limb, then necrosis will occur in the tissues whose vascular demands are highest. In the limbs, these are the muscle bellies, and the lesion produced is Volkmann's contracture.

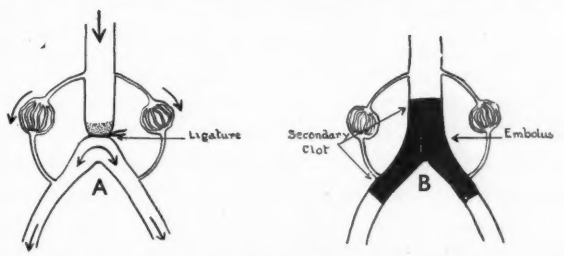


FIG. 156.—A, Effects of ligature, leaving collateral circulation free; B, Mechanical blocking of the collateral circulation by an embolus at the bifurcation of an artery, such as occurred in the first of the three post-embolic ischæmic contractures. (*Modified from Jefferson's diagram*, 1934, *and reproduced by kind permission of Professor Geoffrey Jefferson.*)

In our three cases after embolism the collateral circulation was demonstrably obstructed. In the first, the clot itself occluded the radial and ulnar arteries and thus the collaterals around the elbow in a way simple ligature would not (*Fig.* 156). In the second, where clot had been present seventeen hours, the posterior tibial circulation was never fully restored. In the third, previous embolisms had obstructed the collaterals—the profunda femoris visibly. In injury, however, such a simple mechanical explanation is not present, and the collateral circulation is obstructed by local sympathetic reflexes, arising in the damaged artery, which lead to spasm of the main vessel and of its collaterals (Leriche, 1928).

I have seen this spasm in arterial injuries three times. The first case was in 1937, in a boy of seven upon whom I had reduced a supracondylar fracture of the right humerus. Before reduction the hand and forearm were normal, but immediately

* Schloffer (1901), Burrows (1918), Makins (1919), Métivet (1933).

after the manipulation they changed dramatically, becoming suddenly blanched, cold, anæsthetic, and pulseless. They remained in this state despite six hours of conservative treatment. I therefore explored the arm, and found a small hæmatoma, no larger than a millet seed, in the anterior wall of the brachial artery at the site

FIG. 157.—A hæmatoma in the wall of a brachial artery after reduction of a supracondylar fracture. The patient showed complete arrest of the circulation in the limb.

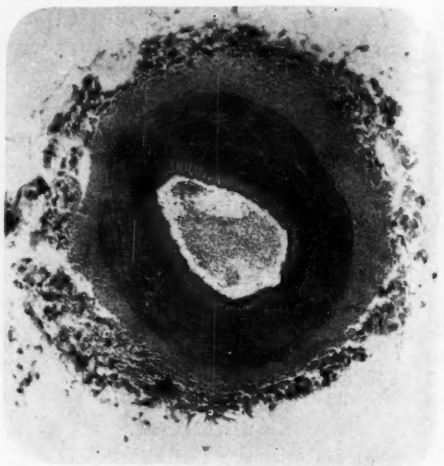


FIG. 158.—The normal brachial artery 1 cm. above the lesion shown in *Fig.* 157.

of fracture. Despite the smallness of this hæmatoma, the whole artery and its radial and ulnar branches were collapsed and pulseless from the insertion of the coracobrachialis downwards. The collateral vessels were equally collapsed—the anterior ulnar recurrent was the size of a retinal artery. Mobilizing and stripping the brachial artery produced no change, but after resection of the damaged portion the arm made a complete recovery. Circulation was fully restored, and there was neither gangrene nor contracture. *Figs.* 157, 158 show the damaged artery and the normal vessel immediately above the hæmatoma.

FIG. 159.—Transverse section of brachial artery in a case of ischæmia of the upper limb due to localized intramural rupture.

A similar happy result followed excision of the damaged portion of the brachial artery in a man of forty-five, in June, 1938. His arm had been crushed in a machine five hours previously. There was no fracture, but the arm was completely ischæmic, paralysed, and pulseless. His artery showed a very localized intramural rupture (*Fig.* 159), but the vessel and its collaterals were pulseless from the subclavian downwards.

In the third case, a boy of seven, a supracondylar fracture of the right humerus produced absence of the radial pulse, which was not restored by manipulation of

the fracture. Exploration took place seven hours after the injury. *Fig.* 160 shows the stages of the operation. The brachial artery was found to be kinked by the upper fragment over a small hæmatoma which was localized to the actual site of fracture where the artery was trapped. The main vessel and the radial and

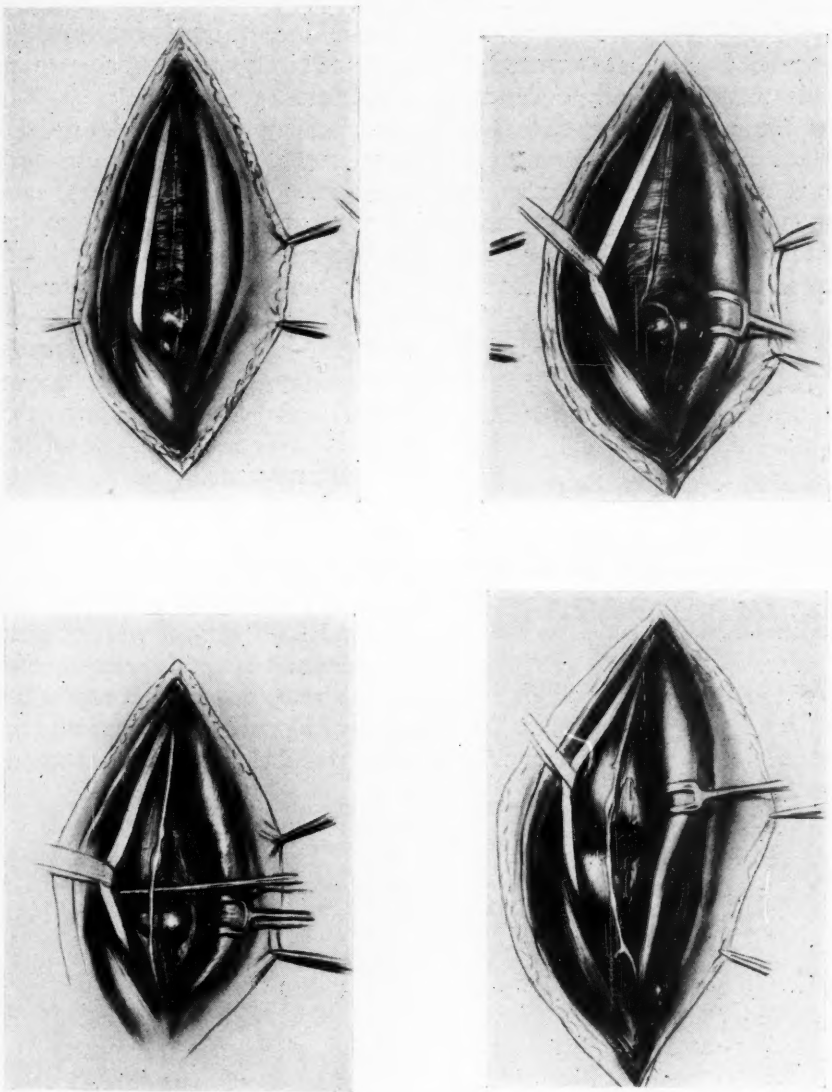


FIG. 160.—Four stages in the liberation of an injured brachial artery seven hours after a supracondylar fracture. The limb showed a complete circulatory arrest which was relieved after liberation of the artery and papaverine injection.

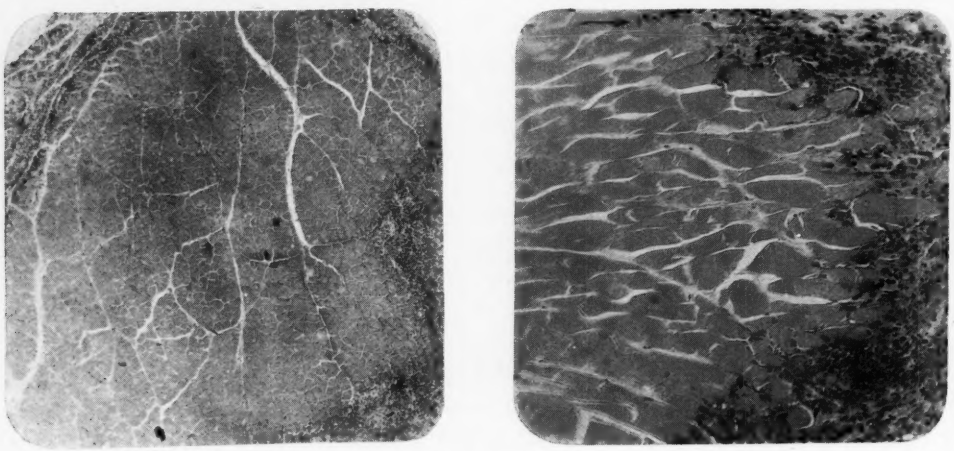
ulnar branches were collapsed from the axilla downwards by an irregular spasm, but there was no localized damage to the artery, and mobilization of it, together with intravenous papaverine injection, succeeded in restoring the circulation and the pulse. Again, no contracture developed.

Thus in these three patients a localized arterial lesion produced a widespread spasm which threatened the very life of the limb. In each instance the spasm was relieved within eight hours, twice after arteriectomy, but it is important to note that Aurousseau (1938), who did the same operation on a similar case at the twenty-third hour, saw his patient develop limitation of extension of the fingers, while Fèvre and Bertrand (1935), Funck-Brentano (1935), Pouzet and Leclerc (1937), and Leveuf (1938), intervening on exactly similar cases at the twenty-fourth, forty-sixth, seventieth, and seventy-second hour respectively, were unable to prevent the development of severe Volkmann's contractures.

The Experimental Production of Volkmann's Contracture.—Additional proof that high-grade arterial and collateral obstruction produces Volkmann's contracture is now available from experimental work. Von Nario (1925) claims to have produced the condition by the injection of 40 per cent salicylate into the dog's axillary artery, ligature of that vessel, tight bandaging for forty-eight hours, and then immobilization of the limb for twenty days; but this bizarre experiment hardly resembles clinical conditions. W. C. Wilson (1902) and Brooks, Kirtley, and Johnson (1934) have, however, produced undescribed ' contractures ' in rabbits by arterial ligation, which seems a more rational procedure.

FIG. 161.—Rabbit showing ischæmic contracture of the right hind limb three weeks after ligature of the ipsilateral common iliac and common femoral arteries.

Working upon a small series of rabbits, I have been quite unable to produce the Volkmann syndrome by the prolonged application of tourniquets to the hind limb with or without ligature of the common femoral artery. Similarly, ligature of the femoral artery with or without the vein, but without any tourniquet, has been equally ineffective. Any combination of ligatures applied to any or all of the external iliac, common femoral, superficial femoral, and popliteal arteries has



A B

FIG. 162.—Histology of ischæmic contracture in the rabbit quadriceps following ligature of the common iliac and common femoral arteries. Lesion six weeks old. A, Low-power; B, High-power. Compare with *Figs.* 163, 155, and contrast with *Fig.* 151.

also failed, as the collateral circulation by the sciatic route is free. If, however, the ipsilateral common iliac and common femoral arteries were tied simultaneously, thus obstructing the main vessel and the collaterals, contracture of the Volkmann type occurred. I have only done this operation on ten rabbits, five of which died, but the survivors all showed an immediate flaccid paralysis, which gradually recovered, but which was superseded after forty-eight hours or so by a rapidly developing flexion contracture. The contracture was well marked after the seventh day. It always affected the knee severely, and in four cases produced as well a marked flexion contracture of the ankle with clawing of the toes (*Fig.* 161). After about fourteen days it is impossible to pull these legs fully straight, and the histology of the affected muscle shows the typical massive necrosis with fibrous tissue 'involucrum' and peripheral round-cell infiltration (*Fig.* 162).

TREATMENT

Prophylaxis.—The belief that Volkmann's contracture is due to arterial injury and reflex collateral spasm has a vital bearing upon the most important part of the treatment of the condition—prevention. Let it be stressed at the onset that preventive treatment must be early, since the fate of the infarcted muscle is decided in hours and not days. Bearing that in mind, preventive treatment must be directed to the relief of the arterial circulation, and must therefore be determined by clinical evidence of arterial occlusion. Any case showing an absent or weakened radial pulse, or weakened oscillations, following a limb fracture, is in danger of the Volkmann syndrome, and it is to this type of case that the term 'threatened Volkmann contracture' must apply. Painful flexed fingers, with acute pain in the forearm on attempting passive extension, do not foretell a contracture, for by the time these appear the necrosis is established and prevention is out of the question.

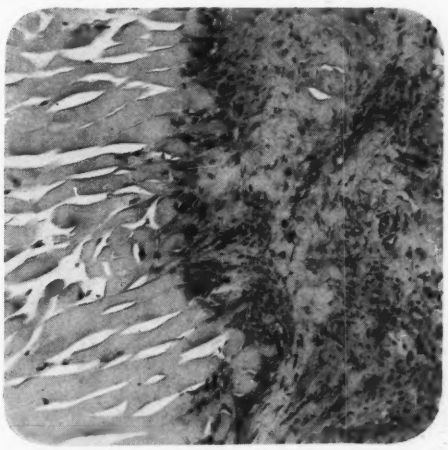


FIG. 163.—Flexor muscle from a five-months old ischæmic contracture after supracondylar fracture of the humerus. Same case as *Figs.* 148, 154; compare with *Fig.* 162.

Taking as a typical case a supracondylar fracture with signs of arterial occlusion, one must first relieve any accessory or extrinsic obstruction, especially *acute flexion* of the elbow. This has been long recognized as a dangerous position, for such acute flexion will obliterate the radial pulse in a normal limb. Similarly, any tight bandage, splint, or plaster which may be present can only increase circulatory embarrassment, while the maintenance of pressure on the artery by persistence of forward displacement of the lower end of the upper fragment cannot but hinder recovery of the circulation. Indeed, the incidence of such mal-union in Volkmann's contracture is at least 40 per cent (Meyerding, 1936), and was 52 per cent in my series (*Figs.* 164, 165).

Accordingly the fracture should be gently reduced by manipulation and, although this has never yet been reported, such a manipulative reduction *may*

release a trapped artery and restore the circulation. Bearing in mind, however, that the first symptom of the arterial block dates more often from a manipulation than from the fracture itself, all such intervention must be gentle and should not be persisted with if reduction is not easily obtained. Gradual reduction by skeletal or other traction is not advisable because of the time which will be lost should it fail, and open reduction without exploration of the vessels has even less to recommend it.

If the fracture has been reduced or is irreducible and the radial pulse remains absent, one must be guided by the circulation in the hand. If this is demonstrably intact, with a pink, warm hand, a rapid circulatory return, and satisfactory oscillations, the arm should be slung at the right angle and watched. If this happy state of affairs is not present, the arm should be operated upon forthwith.

In the first place, the deep fascia of the antecubital fossa and forearm must be widely opened, and the brachial, radial, and ulnar arteries freed and explored. Apart from the deliberate exploration of the vessels, this procedure is the 'aponeurotomy' first suggested by Bardenheuer (1911) and urged by Murphy (1914), Jepson (1926), and the other protagonists of the venous occlusion theory as a means of liberating the subfascial hæmatoma and freeing the veins. Contrary to their hopes, however, an operation limited to this measure will not prevent Volkmann's contracture unless the arterial circulation is fortuitously or deliberately released by the surgeon, which may happen.* If the artery is lacerated or trapped

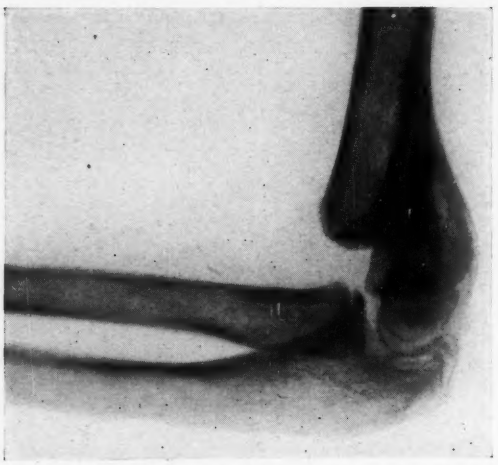


FIG. 164.—Mal-union of a supracondylar fracture of the humerus associated with ischæmic contracture. Same case as in *Figs.* 148, 154, 163, and 165.

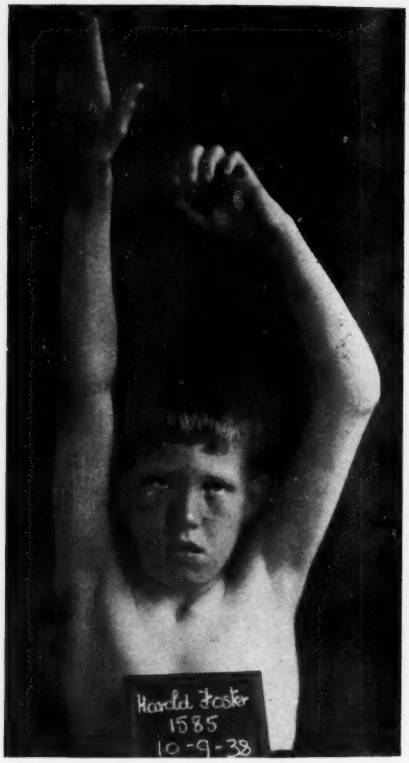


FIG. 165.—Clinical photograph of the patient referred to in *Figs.* 148, 154, 163, and 164.

* In 1938 I explored a femoral artery which had remained pulseless and in spasm eighteen hours after the removal of a tourniquet which had been present six hours. Mere division of the fascial roof of Hunter's canal and mobilization of the artery restored pulsation. There was no hæmatoma and no local arterial laceration.

between the fragments, or if the collateral spasm is at all severe, something more will certainly be necessary, so the artery must be explored every time.

Table VI.—Attempted Prevention of Ischæmic Contracture by Arterial Surgery

Operator	Year	Interval between Injury and Operation	Operation Performed	Result
Cases following Supracondylar Fractures :—				
Plewes	1939	1 hour	Arteriectomy	Complete success
Griffiths	1937	6 hours	Arteriectomy	Complete success
Griffiths	1939	7 hours	Mobilization and papaverine injection	Complete success
Aurousseau	1938	23 hours	Arteriectomy	Limited extension of fingers
Fèvre and Bertrand	1935	24 hours	Arteriectomy	Complete failure
Funck-Brantano	1935	46 hours	Arteriectomy	Complete failure
Pouzet and Leclerc	1937	70 hours	Arteriectomy	Complete failure
Leveuf	1938	72 hours	Arteriectomy	Complete failure
Case following Crush Injury of Upper Arm :—				
Griffiths	1938	5 hours	Arteriectomy	Complete success

It may be that release of an incarcerated or obstructed artery may restore the circulation (Fontaine and Branzen, 1937; P. D. Wilson, 1933); but if, as in my three cases, pulsation is not restored by such release, further steps must be taken. If the artery is not actually damaged or thrombosed, but merely shut down by spasm, restoration of pulsation may, as in my third case, follow the intravenous injection of papaverine. If this fails, or if there is any *localized* contusion or laceration of the artery, the affected portion should be excised between ligatures, the greatest possible care being taken to preserve collaterals.

This operation, arteriectomy, was apparently introduced by Tixier (1907), forgotten, and re-introduced by Leriche, who, with Fontaine and Dupertuis (1937), has made an extensive clinical and experimental study of it. He claims that excision of a length of damaged artery abolishes sympathetic reflexes by cutting off the stimuli, and thus relieves the spasm of the collaterals. The operation, as I have said, gave an excellent result in two of my cases, and I am indebted to Mr. L. Plewes (1940, personal communication) for an account of a similar success in a case of laceration of the brachial artery with ischæmic signs, one hour after the receipt of a compound supracondylar fracture in a child. It cannot, however, be over-emphasized that Plewes operated after one hour, and I after six and five hours with success; while Aurousseau obtained partial success after twenty-three hours. All reported arteriectomies performed later than this have been complete failures. (*Table VI.*)

At the close of the operation, whatever has been done, the fracture should be gently reduced, the wound closed, and the arm slung at a right angle. Further flexion may be allowed after seven days or so.

Treatment of the Established Contracture.—Of treatment of the established contracture I have little to say. In our hands the method of gradual stretching and splinting described by Robert Jones (1907) has given excellent results in moderate and mild cases, while nothing has given really good

results in severe cases. Gradual elastic traction in the apparatus attributed by Böhler (1935) and Kappis (1938) to Mommsen has been used as an alternative method, but the results have not been brilliant.

Operation is indicated, following general surgical principles, for unrecovered nerve palsy, especially of the median. Of fourteen nerve palsies we have had to explore eight. One required secondary suture. Apart from these nerve-freeing operations, the present series of cases has not given enough operative experience to justify dogmatism. I may, however, say that we have found the 'slide' operation of Max Page* (1923) particularly useful in the correction of elbow contracture and of the crippling pronation contracture of the forearm. The operation should be done radically and may have to include anterior capsulotomy of the elbow and complete transection of the pronator teres. It has been done six times in Professor Platt's clinics.

I have no experience of tenotomy (Volkmann, 1881), partial myotomy (Davies-Colley, 1898), " plastic myotomy " (Putti, 1938), tendon lengthening (Page, 1900†), tendon substitution (Kausch, 1904), shortening of the radius and ulna (Garré, 1905), or resection of the metacarpal heads (Steindler, 1936), but the variety and number of operations suggested must indicate how unsatisfactory they are. The only bone operation which we have practised has been resection of the carpus. It has given stable pseudarthroses in good positions, which, though the correction of the fingers is not good, allow the patients to make the most possible use of their hands.

PROGNOSIS

The prognosis in established Volkmann's contracture depends mainly upon the severity of the case. A severe case, with contracture of fingers, wrist, radio-ulnar joints, and elbow, especially if nerve palsy be added to this, will always give a poor result. A great deal depends upon the nerve palsy, recovery of which is essential for the return of any power, but even if this recovers some cases will have so useless, unsightly, and painful hands that amputation may occasionally be indicated. Even in the best cases the hand and forearm seldom become absolutely normal. Growth is retarded, the skin remains thin, and some degree of contracture is apt to persist.

On the whole the outlook is poor. However treated, whatever skill and patience be shown, Volkmann's contracture is a tragedy. Any unsightly and crippling condition in a young limb is a grave enough misfortune, but when, as I hope I have shown, nothing more than careful clinical examination and simple operations will prevent it, the lesion must represent a severe stigma upon modern traumatic surgery.

SUMMARY AND CONCLUSIONS

Based upon observations derived from 32 cases, evidence is produced in support of the theory that Volkmann's ischæmic contracture is due to arterial injury and to the accompanying spasm of the collateral circulation. This view is supported by clinical signs of arterial occlusion in acute and chronic cases. The histology is shown to be that of a muscle infarction. The theory that the contrac-

* But *see also* J. B. Murphy (1912).

† Priority for this operation was later claimed by Littlewood (1902).

ture results from venous occlusion is rejected in view of the absence of any venous compression by hæmatoma in acute cases, the discovery of arterial lesions in operations at all stages, the occurrence of the contracture after arterial embolectomy, and the production of the lesion in a small series of rabbits by arterial ligature.

The early diagnosis of the lesion is discussed, and its prevention by operations designed to relieve arterial obstruction and collateral spasm is described.

Acknowledgements.—It remains for me to offer my sincerest thanks to Professor Harry Platt, whose sage experience and unfailing helpful criticism has given me great encouragement, and to Professor Geoffrey Jefferson in no less measure, especially in connexion with the work on embolisms. Professor John Morley has kindly granted me full facilities for experimental work, Professor S. L. Baker has helped with the histology, and Mrs. K. H. Watkins has given invaluable assistance with translations from German and Italian. To them, as well as to my colleagues, H. O. Clarke and R. Barnes, who have been associated with me in this work, I am very grateful.

The drawings are the work of Miss Dorothy Davidson, and the photomicrographs of Mr. H. C. Taylor. The papaverine preparation used in the case reported was Eupaverine, kindly supplied to me by Messrs. E. Merck (Darmstadt).

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THE TREATMENT, COMPLICATIONS, AND LATE RESULTS OF ACUTE HÆMATOGENOUS OSTEOMYELITIS

BASED ON A STUDY OF 500 CASES ADMITTED TO THE LONDON HOSPITAL DURING THE YEARS 1919–37*

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This review includes 500 cases of acute hæmatogenous osteomyelitis admitted to the London Hospital during the years 1919–37 inclusive. All the bones of the skeleton are included except those of the skull, vertebræ, and phalanges.

Incidence.—Acute osteomyelitis is a diminishing disease in the East End of London. Between the years 1919 and 1928 344 cases were admitted, whereas from 1929 to 1937 there were only 156 admissions. This fall is commonly attributed to the better health and home conditions which now prevail, no other cause having as yet been found.

In this series the usual predominance of boys (67 per cent) to girls (33 per cent) was maintained, the higher incidence in males being attributed to their more adventurous life and to their greater exposure to trauma. *Table I* shows in detail the numbers occurring in each decade, together with the mortality.

Table I.—Age Incidence

Males

Age	*Total*	*Alive*	*Dead*	*Percentage Deaths*	*Percentage to Total Males*
0–9	112	79	33	29	34
10–19	195	152	43	22	58
20–29	15	12	3		
30–39	7	5	2		
40–49	3	2	1		
50–	3	2	1		
Totals	335	252	83		

Females

Age	*Total*	*Alive*	*Dead*	*Percentage Deaths*	*Percentage to Total Females*
0–9	80	51	29	36	48
10–19	66	52	14	21	40
20–29	10	10	0		
30–39	5	5	0		
40–49	3	3	0		
50–	1	0	1		
Totals	165	121	44		

Age Incidence.—The graph (*Fig.* 166) shows the years when the disease was most prevalent, and it represents the total incidence of both sexes in each year of life. Ninety per cent were under the age of twenty and the highest incidence came

* Based on a Hunterian Lecture delivered at the Royal College of Surgeons on January 26, 1940.

between the years of eight to fourteen, comprising 251 cases. The mortality curve shown shaded follows that of the incidence closely, except in the case of children under the age of one year, in whom the death-rate rose to 80 per cent.

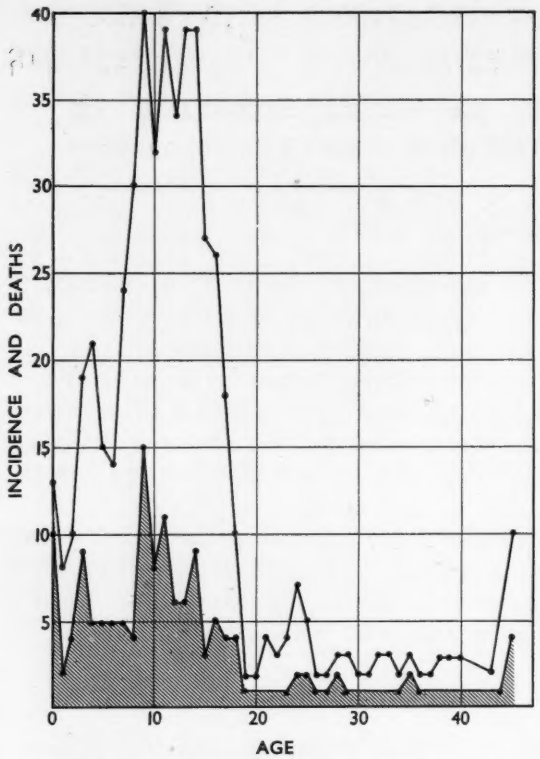

Fig. 166.—Chart of total incidence and total deaths.

Trauma.—There was a history of injury in 43 per cent of cases. Injury was also an important factor in adults, occurring in 17 out of 45 cases. In no case did osteomyelitis supervene on a simple fracture.

Primary Septic Focus.—A primary septic focus was only noted in 93 cases. This figure is low because the importance of a septic focus was not realized in the past, and therefore its presence or absence was rarely placed on record.

In the last three years we have instituted a more careful search and have found a much higher percentage of positive results, 22 out of 49 cases.

In a recent paper in *The Lancet*, Dr. Valentine and I suggested that these foci rather than the infected bone may sometimes be held responsible for the maintenance of the bacteriæmia. I quote the following two cases in support of this view.

Case 1.—A girl, aged 12, was admitted to hospital with an eight days' history of increasing delirium and pain in the arm. On examination she had signs of osteomyelitis of the humerus and pericarditis, and died the next day. Her blood-culture was 1000 colonies of staphylococci per c.c. of blood. She had a septic lesion on one finger, and after death Dr. Gilmour made serial sections of this and found two small veins full of thrombus and containing numerous cocci.

Case 2.—A man, aged 60, died five days after the onset of acute staphylococcal bacteriæmia. He had no bony lesion, but a small infected blister on his heel with an underlying pus pocket. Post-mortem examination showed death from pyæmia, and a microscopical examination of the tissues under his heel again showed small veins full of infected thrombus.

If this theory is a correct one, then clinical improvement should sometimes be noted if these foci could be removed by surgical means, as the source of the bacteriæmia might be checked. I have only done this as yet in two cases, but I think their notes are of interest.

Case 1.—A girl, aged 12, was admitted with five days' history of pain in the left thigh. She showed signs of osteomyelitis of the upper third of the femur and her blood-culture was positive up to 170 colonies of staphylococci per c.c.; the prognosis was accordingly very grave. Surgical treatment consisted in immobilization and aspiration of a subperiosteal abscess. Five days later her blood-culture was still 100 colonies per c.c. and I excised her primary lesion, which was an infected abrasion on the nose. Four days later her blood-culture was negative, although later radiographs showed involvement of nearly all the shaft of the femur. She did well and was transferred to the country.

Case 2.—A married woman, aged 24, was admitted with a healed breast abscess and signs of osteomyelitis of the ilium. The abscess had been opened four weeks previously and she had had pain in the back for two weeks. Her blood-culture was positive up to 100 colonies of staphylococci per c.c. Six days after admission, as her bacteriæmia was still high, I excised the whole of the left half of the breast, including the site of the abscess. Seven days later her blood-culture was negative, although later radiographs showed involvement of one-half of the pelvis, including the hip-joint.

I shall refer again to this form of treatment in a later section.

Radiography in Diagnosis.—Only 31 cases showed X-ray changes on admission, and 17 of these were in adults in whom the disease often has a subacute onset. Definite radiological changes in the affected bone are rarely seen until the end of the second week of the disease, and I think it is correct to say that radiology has little to offer in the diagnosis of acute osteomyelitis.

Bacteriology.—The figures in *Table II* indicate that for practical purposes the staphylococcus may be considered the causative organism of osteomyelitis. The staphylococcus was also responsible for 77 out of the 83 deaths in which the organism was identified.

Table II.—BACTERIOLOGY

Total Cases		*Deaths*	
Staph. aureus	342 (83 per cent)	*Staph. aureus*	73 (87 per cent)
Staph. albus	22 (5 per cent)	*Staph. albus*	4
Streptococci	45 (11 per cent)	Streptococci	3
Anaerobes	2	Other organisms	4
B. coli	1	Unknown	43
Pneumococcus	1		
Unknown	87		

Blood-cultures:—

		Alive	*Dead*
Positive	123	59	64 (52 per cent)
Negative	122	119	3 (2 per cent)

Blood-cultures were unfortunately not carried out on any settled plan and the tabulated results are statistically untrustworthy, suggesting as they do that 50 per cent of cases with a positive culture died. In the past cultures were often not taken unless the patient was very ill, and only one culture was taken, generally a day or so after admission. I am convinced that routine repeated cultures would have given a higher percentage of positive results in the milder cases. Lately it has been our custom to take a culture immediately on admission, again after operation, and later at regular intervals in order, if possible, to correlate the patient's clinical improvement with the disappearance of organisms from the blood. We have found that two clinical manifestations seem to appear when the bacteriæmia stops and the immunity of the serum rises: The patient's appetite returns and the temperature alters from a continuous one to that of a swinging type. There is generally no decrease in the pulse-rate until several days later.

The blood-cultures, to be of any prognostic value, must be quantitative ones, indicating the number of colonies growing in a plate in which 1 c.c. of the patient's blood is incorporated. In cultures from fatal cases the count may go up to hundreds or thousands of colonies, and with a count of over fifty colonies the prognosis is grave. Counts of five to fifteen seem to be common in serious but non-fatal cases.

A persistent or increasing bacteriæmia, indicating that organisms are being thrown off into the blood-stream from some infected thrombus, is of grave significance. The site of this thrombus may be the original septic focus, the infected bone, or some other pyæmic focus elsewhere in the body. We do not believe that the actual multiplication of organisms in the blood-stream occurs except perhaps shortly before death.

Table III.—Illustrating the Value of Quantitative Blood-cultures in Staphylococcal Osteomyelitis

Case	Age	Sex	Site	Days of Disease Culture was Taken	No. of Colonies per c.c.	Result
1	12	M.	Femur	3	900	Died
2	13	F.	Humerus	5	800	Died
3	13	M.	Tibia	6	2	
				10	5	
				14	14	Died
4	14	M.	Tibia	10	4	
				12	2	Lived
5	12	M.	Pelvis	5	20	
				9	12	
				14	0	Lived
6	13	F.	Femur	3	170	
				7	26	
				9	2	
				12	0	Lived
7	Adult	F.	Pelvis	14	6	
				17	45	
				19	110	
				21	22	
				25	9	
				33	0	Lived

Table III illustrates the value of quantitative blood-cultures in assessing the prognosis of this disease. It is interesting that the only patients who have survived a count of over 100 colonies per c.c. are those who had their primary lesion removed by surgery (*Cases* 6, 7). *Case* 3 shows the importance of an increasing colony count, whatever the degree of bacteriæmia, as a guide to the prognosis.

Mortality.—Death occurred in 127 cases (25 per cent) and the rate was the same in the two sexes. Reference to *Table IV* shows that the death-rate remained almost constant in consecutive hundred cases, despite the decreased incidence and conservative treatment of recent years.

Table IV.—The Mortality per Hundred Cases

First hundred	(1919–21)	31
Second hundred	(1922–24)	28
Third hundred	(1925–27)	19
Fourth hundred	(1928–31)	23
Fifth hundred	(1932–37)	26

The next chart (*Fig.* 167) illustrates the numbers of cases which died on each day of the disease.

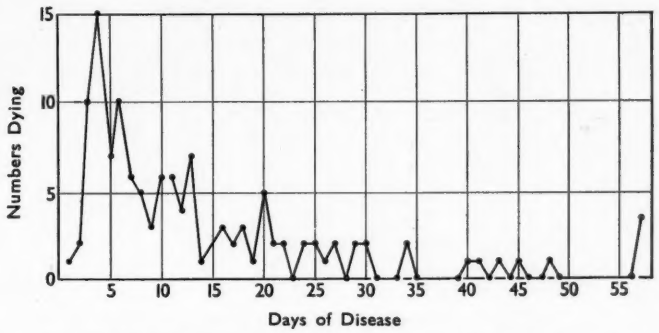

Fig. 167.—Chart illustrating the days of disease when death occurred.

Forty-five cases, or over 34 per cent, died in the first week, and over 66 per cent in the first fourteen days. The patients who died in the first week did so from an acute bacteriæmia against which surgery was of no avail. It seems evident that the mortality of osteomyelitis will continue to be high until methods are found to conquer staphylococcal blood-stream infection.

Table V.—POST-MORTEM FINDINGS IN 92 CASES

	Total	*Percentage*
Pyæmia	89	97
Anæsthetic death	2	2
Bronchopneumonia	1	1
Local arthritis	34	37
Pyæmic arthritis	14	15
Ante-mortem clot	28	30
Lung abscesses	75	81
Myocardial abscesses	41	46
Endocarditis	10	11
Other abscesses	50	54
Other bone	14	15

The next table (*Table V*) shows the post-mortem findings on 92 cases. These post-mortems were all performed in the pathological institute at the London Hospital and I am grateful to Professor Turnbull for allowing me to publish them. There were 89 deaths due to pyeæmia, i.e., multiple macroscopical abscesses throughout the body. These figures show clearly that death in this disease is almost always due to blood-stream infection and its complicating abscesses. Death from toxæmia alone, as described in some text-books, does not occur as a practical reality.

Bones Affected.—*Table VI* shows the incidence in the different bones and the resulting mortality.

Osteomyelitis of the pelvis runs a different course from infection of the long bones because of its anatomical structure. The child's pelvis has many epiphyses, little marrow cavity, and a good blood-supply. It follows that there are many sites in which infection may start, that the intense pain of an intramedullary infection may be absent, and massive destruction of bone is rare.

Unfortunately the anterior inferior iliac spine is the commonest site for infection, and involvement of the hip-joint is then almost certain. Also the disease often spreads across the pelvis to invade the opposite side; this process may be relatively slow, and its course can be followed by repeated X-ray examinations

Table VI.—Bones Affected and Their Mortality

Bones	*Total*	*Alive*	*Dead*	*Percentage*	*Mortality Percentage*
Pelvis	39	22	17	8·0	44
Femur, Upper	43	22	21	9·0	49
Middle	11	7	4	2·0	36
Lower	73	49	24	14·0	33
Tibia, Upper	82	61	21	16·0	26
Middle	25	16	9	5·0	36
Lower	54	45	9	11·0	17
Fibula	39	33	6	8·0	15
Patella	2	2	0	0·4	
Os calcis	19	15	4	4·0	21
Tarsus	11	10	1	2·0	9
Metatarsus	8	8	0	2·0	
Sternum	1	0	1	0·2	
Clavicle	7	7	0	1·0	
Scapula	5	4	1	1·0	
Humerus, Upper	24	19	5	5·0	21
Middle	14	14	0	3·0	
Lower	6	6	0	1·0	
Radius	18	17	1	4·0	6
Ulna	16	13	3	3·0	19
Carpus	1	1	0	0·2	
Metacarpus	5	5	0	1·0	

The total femurs equal 25 per cent of all cases, with 38 per cent mortality.
The total tibias equal 32 per cent of all cases, with 24 per cent mortality.

Fig. 168.—Radiograph in the case of a boy who developed bilateral osteomyelitis of the pelvis with involvement of both hips. This disease started in the anterior inferior iliac spine. He recovered and was transferred to an orthopædic hospital in the country.

Fig. 169.—Radiograph illustrating a typical after-result of osteomyelitis of the pelvis which has involved the hip-joint.

(*Fig.* 168). The ultimate result is generally asymmetry of the pelvis and bony ankylosis of the hip (*Fig.* 169). If the disease is subacute in type abscesses only form very slowly, and this type may be mistaken for a rapid form of tuberculous infection; a positive blood-culture in the earlier stages and a high staphylococcal antibody content in the blood are then aids to diagnosis.

In adults osteomyelitis of the femur generally attacks the shaft, and one type is especially worthy of notice. The lesion appears on the inner aspect of the

bone, and the infection instead of spreading around the shaft advances up and down, leaving about one-third of the circumference unaffected (*Fig.* 170). The reason for this may be the firm attachment of the periosteum to the linea aspera. Pathological fractures are especially liable to occur in this type.

Table VII.—PRIMARY OPERATIONS

	Total	*Alive*	*Dead*	*Percentage Deaths*
Periosteal abscess drained	117	93	24	21
Periosteum incised and bone drilled	41	33	8	20
Medullary cavity drained	280	220	60	22
Primary amputation	2	2	0	
Diaphysectomy	9	7	2	
Joint drainage	20	7	13	65
Sequestrectomy	6	6	0	
No operation	31	5	26	84

Cases Needing Sequestrectomy :—

Drainage of medullary cavity = 38 per cent of cases living
Drainage of periosteal abscess = 29 per cent of cases living
Drilling of bone = 21 per cent of cases living

FIG. 170.—Radiograph showing an infection of the shaft of the femur in an adult. The patient recovered and a pathological fracture did not occur.

Primary Operations.—Four hundred and sixty-nine cases had operations on admission or shortly after. The various operations performed and their mortality are shown in *Table VII.* The death-rate of the three main types of operation were the same, 21 per cent, although it is not perhaps fair to compare those drilled with the other two groups, since the figures are so much smaller. It would appear, therefore, that the mortality tends to be the same whatever the operation employed; this would fit in with the bacteriological and post-mortem findings in the fatal cases. Immediate amputation saved the lives of the two cases on whom it was performed, but they both died later from recurrent infections in other bones and pyæmia. This suggests that amputation should not be performed until the patient has acquired a real or artificial immunity against the staphylococcus. Diaphysectomy was only done for infections of the fibula and first metatarsal; the late results of this operation are dealt with subsequently. Primary joint drainage carried a high mortality. Acute infection of the upper end of the femur often presents as an arthritis of the hip, and in many cases the bone infection was only discovered later or at post-mortem examination.

Twenty-six of the 31 cases which had no operation either had fulminating bacteriæmia or were only diagnosed in the post-mortem room. This group accounted for 22 per cent of all the deaths, and it is unlikely that surgery, however early, would have benefited them, since they were killed by their blood infection.

This table also shows that the incidence of sequestrectomy was lowest after drilling the bone. It is reasonable to assume that the lessening of intramedullary tension followed by complete rest in plaster limited the destruction of bone in this group.

Table VIII.—SECONDARY OPERATIONS

	Total	*Percentage**	*Alive*	*Dead*
Sequestrectomy	120	24	120	0
Amputation	23	5	18	5
On other bones	43	12	38	5
Joint drainage	22	24	17	5

* The percentages in the above table refer to the grand total of 500 cases.

Table IX.—COMPLICATIONS

	Total	*Percentage**	*Alive*	*Dead*
Pathological dislocation	5	1·0	5	0
Pathological fracture	6	1·0	6	0
Secondary hæmorrhage	6	1·0	5	1
Nerve lesions	8	1·6	8	0

* The percentages in the above table refer to the grand total of 500 cases.

Secondary Operations (*Table VIII*)—All these except sequestrectomy took place while the patient was first in hospital. The mortality was low, as the patients had usually recovered from their bacteriæmia and had developed a high immunity against the invading organism before the second operation. Amputation was most commonly carried out for suppurative arthritis combined with a severe bony lesion, and the death-rate was surprisingly low when one considers that the operation was performed on children who were recovering from a severe illness. The number of cases developing metastatic bone lesions was small and few died. It is evident that infection of another bone has little bearing on the immediate prognosis, provided that the patient has overcome the bacteriæmia which must have given rise to the secondary lesion. It should be noted that secondary joint drainage was rarely needed, and that its mortality was only 23 per cent as compared with 65 per cent when the joint was drained at the first operation.

COMPLICATIONS

Pathological dislocation occurred once in the knee and four times in the hip. These were usually late manifestations and were not fatal. Pathological fractures were very rare and all united except one which I was unable to trace.

The nerves to be affected were the musculospiral four times, the external popliteal twice, and the posterior interosseus twice. All were post-operative lesions. One musculospiral lesion was treated with secondary suture and did well. The external popliteal lesions were permanent, but the remainder recovered without surgical intervention. (*Table IX.*)

Suppurative Arthritis.—Acute arthritis was only included in this series if the organism had been identified in the exudate or if later radiographs showed bony changes in the affected joints.

Table X.—ACUTE ARTHRITIS

(118 cases, 24 per cent incidence)

	Total	*Alive*	*Dead*
Local arthritis	110	75	35
Pyæmic arthritis	8	4	4

Mortality with arthritis 32 per cent

Mortality without arthritis 24 per cent

Immediate Results of Treatment:—

	Total	*Good*	*Limited*	*Ankylosis*	*Amputations*	*Dislocations*	*Died*	*No Trace*
Aspiration	56	8	7	12	8	0	14	10
Drainage	48	3	3	9	9	0	18	7
Nil done	14	0	0	7	0	2	3	2

Table X shows that the incidence of arthritis need not be looked upon as one of grave significance, since the effect on the immediate prognosis was moderate. Acute arthritis had, however, a very bad effect on the ultimate function of the limb. Only 37 per cent of these patients who left hospital had a movable joint; the remainder were permanently crippled. The joints which received no surgical treatment were mostly cases of hip infections which were often only diagnosed by later radiographs; this accounts for the high incidence of ankylosis in this group. The knee was most commonly infected, with a total of 50 cases; the hip was second, with 29 cases; and the ankle third, with 15 cases.

I found that in this series 30 per cent of infections of the long bones and the pelvis were followed by infection of the adjacent joints.

FOLLOW-UP STATISTICS

Out of 500 patients 373 lived, and of those I was able to trace 223, or 59 per cent. Of the 223 cases, 160 were examined in the follow-up department, 50 answered a questionnaire, and 13 had died, giving a late mortality of 3·5 per cent.

The results to the questionnaire and to the examinations were conveniently divided up into three groups: Those whose primary attack had been ten or more years ago (1919–28)—this group were all adults, and their final results could be accurately determined; those whose attack lay between five and ten years ago (1929–33); and those recent cases who were admitted during the last four years (1934–7). The late results of the last group are not comparable to the others as sufficient time has not yet elapsed.

My criteria for a good clinical result were as follows: (1) A completely healed wound; (2) No deformity of bone or limb apart from a scar; (3) No recent active focus in any part of the body. I found that in all groups the percentage of good results was similar, and it was gratifying to find such a high percentage of cases able to carry on with their work. These results are set out in detail in *Table XI*, and they are, I think, self-explanatory.

Table XI.—FOLLOW-UP STATISTICS

	All Groups		*Group 1*		*Group 2*		*Group 3*	
	Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage
At work	195	87	121	89	36	92	38	80
Dead	11	4	8	6	2	5	1	2
Good result	110	49	65	47	20	51	25	53
Recurrent operation	87	39	58	42	18	46	11	23
Recurrent discharge	45	20	22	16	8	20	15	32
Recurrent pain	45	20	32	23	7	19	6	12
Deformity	73	32	48	35	9	23	16	34
Other bone operations	44	19	36	26	4	10	4	9
	223		137 cases		39 cases		47 cases	

In the last group the incidence of secondary bone infections and recurrent operation was naturally lower than in the other groups. It will be instructive to trace these patients over the next ten years to see if the more conservative methods of treatment have any effect in diminishing the incidence of recurrent bone infection. I was interested to find that none of the atypical operations presented a good clinical result (*Table XII*).

Table XII.—Late Results of Types of Operations

	Total	*Good*	*Percentage*
Periosteal abscess drained	52	21	41
Bone drilled	30	19	63
Medullary cavity drained	125	67	53
Primary amputation	2	0	
Primary joint drainage	4	0	
Primary sequestrectomy	4	0	
Nil done	3	2	
Diaphysectomy	3	0	
	223	110	

Both cases receiving immediate amputation died later, as I have already described.

Three patients were examined on whom diaphysectomy had been performed. In the first the centre of the fibula did not re-form and caused a valgus deformity of

Fig. 171.—Radiograph showing non-formation of the shaft of the fibula after diaphysectomy some years previously.

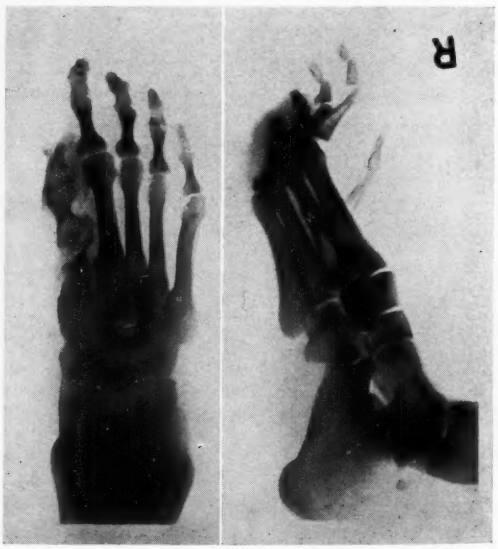

Fig. 172.—Radiograph of the foot, showing the deformity which resulted from diaphysectomy of the first metatarsal.

the ankle (*Fig.* 171). The second patient, another fibula case, had a permanent lesion of the external popliteal. The third had had the shaft of the first metatarsal removed and her deformity is seen in the accompanying radiographs (*Fig.* 172).

Three cases of primary joint drainage came up with ankylosed joints, and the other had died. All cases of primary sequestrectomy had a persistent sinus.

Of those examined, 21 per cent had suffered from suppurative arthritis at the time of their first attack. These results are shown in *Table XIII*; they are very similar to the figures given in a previous table for the immediate results of this complication.

Table XIII.—Follow-up Statistics in Cases with Arthritis

(34 cases, 21 per cent of total of 160 cases in the Follow-up Department)

Good result	6
Ankylosis	17 (50 per cent of joint cases)
Limited movement	11

Results of Treatment:—

	Total	*Ankylosis*	*Limited*	*Good Result*
Aspiration	17	5	8	4
Drainage	7	4	1	2
Nil done	10	8	2	0
	34	17	11	6

In a further 20 cases there was joint limitation from extra-articular adhesions without there being any evidence of actual joint infection. The knee, with 9 cases, was the commonest joint to be involved in this manner.

TREATMENT

In acute osteomyelitis the blood-stream is invaded by staphylococci from a superficial lesion which may be obvious or so small as to pass unrecognized. Some of the cocci settle in a bone and produce a fresh lesion which gives rise to toxæmia and often to further bacteriæmia. The treatment of the acute condition should therefore be directed toward: (1) The toxæmia; (2) The bacteriæmia; and (3) The local lesion. These three factors must be considered in every case of osteomyelitis, but their relative importance varies with the different clinical types of the disease.

1. The Toxæmia.—In a recent paper (Valentine and Butler, *Lancet*, 1939) evidence was given strongly suggesting that the most important antibody in human staphylococcal infection is the antileucocidin. Since toxæmia plays an important part in every case of osteomyelitis it is desirable that antitoxin be given as soon as possible in the majority of cases. The degree of toxæmia depends largely on the specific immunity of the patient; if this is low it should be artificially raised by suitable doses of serum.

In our experience the administration of antistaphylococcal serum by the intravenous route is often accompanied by severe and dangerous reactions, so we are at present only giving it intramuscularly in doses of from 25 to 50 c.c. As a rule we have found that the immunity is either lower than normal or within normal limits, and that it only rises during the second week of the disease. We believe that the injection of serum not only combats the toxæmia but also lessens the danger of further septic thrombosis and fatal bacteriæmia.

The antistaphylococcal sera at present on the market are standardized on the basis of antihæmolysin content. In our opinion this antibody is of doubtful value

in the treatment of staphylococcal infections. Our evidence suggests that the antileucocidin is the important antibody in these sera and in the preparation of the sera used by us particular attention has been paid to the antileucocidin content.

2. The Bacteriæmia.—This when severe may have its source either in the primary focus or in the bone. Repeated quantitative blood-cultures are of the utmost value in demonstrating the degree of bacteriæmia, as the immediate prognosis can be determined by the increase or decrease of the number of organisms in the blood. In severe cases with a high bacteriæmia excision of the primary focus is justifiable in order to eliminate a possible source of infection. Cases illustrating this method of treatment have already been described. The immediate removal of the bony lesion was not successful in this series; but secondary amputation should be seriously considered in patients who develop an increasing bacteriæmia in the second or third week of the disease. These patients have acquired a good antitoxic immunity against the staphylococcus, and removal of the probable source of the bacteriæmia may enable them to overcome successfully their residual systemic infection.

Chemotherapy.—Individual clinical triumphs from the use of drugs of the sulphanilamide group in the treatment of osteomyelitis have been reported from time to time in the literature, but no large series has been published. Our experience of this form of chemotherapy has been disappointing. We have employed sulphanilamide, uleron, and M. & B. 693, but are unable to say that any of them has had any appreciable effect. This lack of success is perhaps not surprising. The available evidence suggests that these new drugs are effective against the hæmolytic streptococci and the pneumococci in virtue of the fact that they hinder the full development of these organisms. In particular the elaboration of surface antigens or capsule is prevented, with the result that the bacteria fall a ready prey to the phagocytes; there is little to suggest that these drugs possess any antitoxic activity. Since *Staph. pyogenes* owes its pathogenic action mainly if not exclusively to the soluble toxins which it produces, it is hardly to be expected that infection due to this micro-organism would be amenable to these drugs.

3. The Local Lesion.—The immediate surgical treatment of acute osteomyelitis consists in immobilization and relief of tension. Immobilization is as important before operation as it is afterwards and plaster-of-Paris is undoubtedly better than splints in this respect, although its use is sometimes impractical before operation as it makes repeated examination of the affected bone impossible. The importance of relieving tension is firstly to relieve pain, secondly to diminish the toxæmia, and thirdly to prevent undue bone destruction.

In cases with severe bacteriæmia and few local signs it is generally best to immobilize the limb and limit surgical procedures to aspiration or drainage of a subperiosteal abscess if present.

These patients are too ill to feel severe local pain, and the bony lesion can be dealt with more radically should this be necessary when the patients' immunity has been raised and the bacteriæmia controlled.

In less severe cases with well-marked signs of intra-osseous tension and toxæmia, free incision of the periosteum, drilling the bone, and fixation in plaster is the method we usually adopt.

Acute arthritis has been shown to be the commonest local complication of this disease and should receive immediate active treatment. Primary drainage of the

joint is inadvisable and in this series carried a high mortality; repeated aspiration and immobilization will generally control the infection, although open drainage may in some cases be needed later in the disease.

Outline of Procedure.—The treatment of every severe case of acute osteomyelitis must be considered as a separate problem, and the following may be taken as an outline of the treatment to be adopted whenever possible:—

On Admission:—

1. Obtain a specimen of blood for quantitative culture and for estimation of the staphylococcal antibody content.
2. In severe cases give 25–30 c.c. of staphylococcal antitoxin (with a high antileucocidin content) intramuscularly.
3. Provide absolute rest to the affected bone.
4. Give a plentiful supply of fluids in the form of glucose lemonade, as this is usually well tolerated.
5. Decide on the local surgical treatment, which may be: (*a*) Aspiration; (*b*) Incision of the periosteum; (*c*) Incision of the periosteum and drilling of the bone.
6. If there is a definite primary septic focus excise it if possible in those cases with a high bacteriæmia.

After Operation:—

1. Obtain repeated blood-cultures in severe cases.
2. Maintain complete rest to the site of operation, preferably in plaster.
3. Keep a careful watch for the onset of local arthritis.
4. Examine daily for possible pyæmic manifestations in other bones, joints, or viscera.
5. Should secondary anæmia become marked, small repeated blood transfusions are useful in the later stages of the illness.

Once the patient has overcome his bacteriæmia the treatment depends on the course of his local lesion, which should be treated according to accepted surgical principles.

SUMMARY

In the first part of this article the incidence, treatment, complications, and mortality of 500 cases of osteomyelitis have been described. It has been shown that the mortality of this disease is due to staphylococcal bacteriæmia and that in this series the immediate prognosis was little affected by the various surgical procedures adopted. The importance of the primary septic focus has been discussed and the type of case which might benefit from excision of the primary lesion indicated. Quantitative blood-cultures have been described and their importance in assessing the immediate prognosis has been emphasized.

In the second part I have presented the results of a follow-up of 223 cases, 137 of which were seen ten or more years after the original infection. From these results it seems evident that about 50 per cent of patients can be expected to make a perfect recovery, but that the remainder will be subject to either deformity, pain, discharge, or recurrent infection. It is to be hoped that more modern surgical methods will better these figures in the future by limiting the degree of bone destruction and by controlling the onset of recurrent inflammation.

In the final section I have dealt with the outline of treatment under three heads: the possibilities of serum therapy to combat the toxæmia have been discussed; excision of the primary and bony foci has been considered in the treatment of the bacteriæmia in conjunction with the information obtained by repeated blood-culture estimations; rest and relief of tension sums up the principles of immediate surgical intervention.

I hope that I have emphasized throughout that the surgical treatment of a serious case of osteomyelitis must entail the active co-operation of a bacteriologist and a surgeon. I should like to end by urging a far closer co-ordination between bacteriology and surgery than has been the custom in the past, so that the high mortality of this and other acute staphylococcal infections may eventually be reduced.

TWO CASES OF NON-MALIGNANT STRICTURE OF THE ŒSOPHAGUS

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AMONG the causes of simple stricture of the œsophagus, the swallowing of corrosives, whether by accident or with suicidal intent, is one of the most common.

By way of treatment a variety of non-operative procedures have been tried, and among them dilatation by Jackson's bougies at regular intervals under the guidance of direct vision through an œsophagoscope, has proved one of the most successful; but in not a few cases the result has been disappointing. The simpler method of passing ordinary gum-elastic bougies blindly at frequent intervals has also had its defeats. The failure of many of these cases to derive lasting benefit by these methods has sometimes led to the adoption of surgical interference, often of great magnitude.

For this reason it is considered by the writer to be of the greatest importance to educate the patient in the self-passage of bougies. Once this method has been learned the bougies can be used as often as it proves necessary without other assistance and without the necessity of visiting hospital. In this way the patient soon feels in command of the situation, and by means without pain or even inconvenience. All the time the patient must be encouraged to take nourishment by the mouth and to get on to semisolid or solid food as soon as ever possible, for each bolus acts as a further dilator. It may take months to restore normal swallowing capacity, and occasional dilatation may have to be kept up for years to counteract the tendency to recurrence, but this is no great hardship in the expectation of eventual cure. The dangers associated with self-dilatation have probably been exaggerated, and it is doubtful if they are greater than those of dilatation under direct vision. It has been suggested that in those cases in which a catastrophe has occurred, post mortem examination has sometimes revealed some unsuspected complicating pathological condition.

The writer has previously recorded a series of cases, several of which were successfully treated by self-dilatation.[1] The value of perseverance in their management is well illustrated by the following account of yet another case which gives further encouragement in the use of the method :—

Case 1.—**Simple stricture of the thoracic oesophagus treated by dilatation, with numerous vicissitudes but with ultimate success; as a result the patient more than doubled her weight.**

The patient (A. P.), a female aged 39, was thoroughly happy in her work and environment until July of 1937, when she swallowed half a teaspoonful of concentrated hydrochloric acid in mistake for Phospherine. She was immediately taken to hospital, where she remained for eight weeks. At first her throat was very sore, and attempts to swallow hurt her very much, but it gradually improved and by the end of her stay she was able to take soft food like eggs and milk, minced fish and potato, but only with some difficulty and a good deal of soreness. From the hospital she was sent to a Convalescent Home for three weeks,

and it was during her last week in residence there that she experienced great difficulty in swallowing the more solid food provided. After returning home the dysphagia steadily got worse, and early in December she had to go back to hospital for treatment. Œsophagoscopy was carried out, but "attempts to dilate the stricture with bougies were not very successful". No effort was made to follow up any slight advantage by the use of bougies, and she continued to lose weight. In February the patient returned to the same hospital, where another œsophagoscopic examination was made and this time some improvement followed. But the betterment proved evanescent, and the dysphagia soon got so troublesome that on April 23, 1938, she was transferred to Hammersmith Hospital under my care.

On admission she was found to be a big woman, but thin, wasted, hollow-eyed, too weak to stand, and exceedingly nervous. Her weight was 6 st. 13 lb., in contrast to 12 st. before the accident. There was no abnormal temperature and the pulse was regular but of poor tension and volume. Blood examination showed 3,600,000 red cells, with hæmoglobin 70 per cent and colour index 0·98. There was no leucocytosis. Blood-pressure, 100/70. She was scarcely able to swallow her own saliva and lay in bed with a full sputum mug at her bedside. Though there was the greatest difficulty in getting even fluids down, she could sometimes swallow bits of hard biscuit, which she said kept her throat open. The patient had some cough, but there were no signs of pulmonary or other visceral disease. There was no localizing sensation on deglutition, and no external physical signs of the œsophageal lesion. An opaque swallow on April 25 showed "an obstructive lesion immediately below the level of the aortic arch (*Fig.* 173), through which even the thinnest medium passed with difficulty. There was slight dilatation proximal to the stricture." The patient was very difficult to manage and resented the suggestion that bougies should be tried. When attempts were made she was entirely non-co-operative, but, nevertheless, on two occasions a very small instrument not more than 2 mm. in diameter was passed, but it was obvious that progress by dilatation was going to be slow and difficult.

FIG. 173.—*Case* 1. The stricture in April, 1938, before commencement of treatment at Hammersmith Hospital.

In view of the serious condition of the patient, it was decided to carry out gastrostomy as a preliminary to further treatment, and this was done on May 3, under gas-oxygen and ether. An ill-considered attempt to pass a bougie was made 48 hours after the operation; this upset the patient very much, and the temperature ran up the same evening to nearly 103° F., but it fortunately came down next day. Three weeks later bougies were resumed, but the patient was terrified, and for long we could not secure her co-operation. With great perseverence and persuasion my assistant, Mr. R. H. Franklin, and my then House Surgeon, Mr. D. D. Steele-Perkins, overcame her fears, and attempts were more successful and she began to make some effort to help.

As her general condition improved treatment became easier, and on one or two occasions a bougie of 4 mm. in diameter was retained in situ for a minute or two. At the end of a month from the time of operation she was swallowing saliva with but little difficulty. Two months later a considerable amount of liquid nourishment was being taken by the mouth, and she could manage some semi-solid food. The report of further X-ray examination on July 2 was as follows: "The constriction previously reported persists. Barium emulsion passed slowly through the stricture, which has a diameter of $\frac{1}{8}$ in. In five minutes about $\frac{1}{2}$ oz. of emulsion has reached the stomach. The upper portion of the stricture is slightly wider than on April 25."

Gradually slightly larger bougies negotiated the stenosis and by early in August she assisted with the daily use of the bougie, was able to take nourishment by mouth, her

weight had gone up to 8 st., and she looked well. During all this time the pulse varied between 90 and 100, and there were outbursts of slight pyrexia. Towards the end of her stay she was up and about and all feeding was by the mouth.

In order to be nearer her own home she was transferred to the hospital where she was originally admitted and where it was hoped the treatment by bougies could be continued. To our great disappointment the patient relapsed and had to be re-admitted to Hammersmith in November, her condition having become so severe that she once more had difficulty in swallowing saliva. It transpired that for purposes of dilatation a small Hurst's weighted bougie had been used, but this treatment had not been checked by X rays and it is highly probable that the stricture was not traversed. The patient had lost weight again and for a time had severe diarrhœa. Occasionally after the use of the bougie she coughed and retched and brought up a drachm or so of pus, and on one occasion she had fainted.

On re-admission on November 3 it was obvious that she had lost ground since August, but was still much better than at the time of her first admission. Her weight was 7 st. 11 lb., blood-count 4,700,000 red cells, and Hb 88 per cent, with no excess of leucocytes. The basal metabolic rate was 14 per cent. The temperature was normal and pulse rather erratic. Occult blood was not present in the stool. The X-ray barium swallow was reported as follows: "Compared with appearances seen in July, 1938, there is now complete œsophageal stenosis, not even the thinnest medium passing through the gullet."

In spite of this report the patient was able to swallow erratically. She could take nothing before midday and had to eject her saliva, but at that time she could slowly drink a cup of milky coffee and by 4 p.m. was able to drink some tea and eat a little bread and butter. She improved a little and in a fortnight gained a few pounds in weight. But her dread of bougies prevented effective treatment and she could not be persuaded to swallow a silk thread.

In view of the standstill condition it was proposed to operate with the object of carrying out retrograde dilatation. In the hope of getting rid of the necessity of using bougies the patient agreed, and the operation was carried out on December 8, 1938. After suitable preparation the abdomen was opened to the right of the gastrostomy, the stomach drawn into the wound and incised near the cardia. A small bougie, 4 mm. in diameter, was easily passed from below up through the stricture, and this was followed by one twice the size. Another yet slightly larger was now passed down the œsophagus from the mouth until it presented in the stomach. A piece of rubber drainage tubing of about a centimetre in diameter was pushed on to the end of the solid bougie and firmly tied, and the bougie was withdrawn up the œsophagus, carrying the rubber tube through the stenosis and leaving about 10 in. projecting from the mouth. The distal end of the rubber tube was firmly anchored to the stomach wall with a suture of No. 3 chromicized catgut, and the opening in the stomach and the abdomen closed. It was intended to leave the rubber tube in situ for three or four days, and it had been so securely fixed to the stomach wall that it seemed highly unlikely that it would work loose or that it could be easily withdrawn. To our amazement the patient managed to pull out the tube on the evening of operation. She had completely recovered from the anæsthetic and it was thought that she could safely be left, but in a fit of uncontrolled impulse the contretemps occurred. An extremely anxious four days followed, with continuous pyrexia of 101° F., but eventually apprehension subsided.

As I was loath to lose the advantage of the dilatation which had been secured with so much pains, I arranged for an œsophagoscopic examination in the hope that a Souttar's tube might be placed in the opened stenosis. Mr. Ivor Griffiths carried out the examination for me on the fourth day under evipan anæsthesia. The œsophagus contained a considerable quantity of watery fluid and stale blood, and vision was obscured. A small Jackson's dilator was passed through the stenosis without difficulty, but the smallest sized intubation tube could not be passed farther than an inch, and could not be safely left in situ.

Despite the derangement of our plans, swallowing was much improved, and about three weeks after operation all nourishment was taken by the mouth, the gastrostomy feeds being discontinued. After the lapse of another week she could take a meal of soft solid food, though all meat had to be minced. To anticipate any tendency to relapse, the use of bougies was again proposed, and the patient was now quite willing to try, and was again instructed with great patience in the self-passage of bougies, which were begun on January 4, 1939. In spite of her good intentions real co-operation was lacking, but as the result of daily

encouragement, both by medical and nursing staff, she eventually learned to pass bougies herself, and with increasing ease.

As a result, she made good progress and was able to leave hospital on February 14, just ten weeks after the retrograde dilatation. By that time the gastrostomy had been allowed to close and she was able to take a sufficiency of light food by the mouth. The patient could pass a bougie, designated size No. 15 (8 mm. in diameter) quite easily, but No. 17 became arrested in the stenosis. Her weight on leaving hospital for this second time was 9 st. 9 lb.

A month later she reported herself as very well, was taking ordinary food with ease and weighed 10 st. 12 lb. By July 15 she had gained further weight and turned the scale at 11 st. 9 lb. and was taking solid food without trouble, and had started work. But in spite of this good clinical progress it was disappointing to receive the following X-ray report: "The œsophageal stricture previously reported persists. It commences just below the aortic arch. Lumen of œsophagus is slightly larger than on February 8. One ounce of barium emulsion of consistence of cream passed down œsophagus in approximately five minutes."

When this patient reported on December 13, that is one year and eight months after her first admission to Hammersmith Hospital, she was well and cheerful and in regular employment as a cashier in a store. She was taking ordinary food and eating at an ordinary rate, like the other members of her family. All lost weight had been regained, for she now turned the scale at 12 st. 4 lb. The bougie size No. 15 passed easily and she was using it twice every day, size No. 17 engaged in the stenosis with some hesitation and was firmly gripped. In April, 1940, she was splendidly well, weighed just over 14 st., and had no difficulty in swallowing. She was still passing the No. 15 bougie daily.

Comment.—The latent period between the injury and the gradual development of the stenosis makes one wonder if the traditional treatment of these œsophageal injuries is on correct lines. Naturally the claims of appetite and desire encourage the patient to persevere in swallowing, but it might be better to provide physiological rest for the injured viscus by an early resort to gastrostomy. Though I have no personal experience of such practice, nor am I aware that it has been tried, I have always been much impressed by the marked improvement that occurs in most œsophageal lesions as the result of gastrostomy alone. In another field—traumatic rupture of the urethra—considerable experience has fully convinced me of the great value of diverting the urine away from the injured area. In dealing with that condition I have not had the courage to rely on diversion alone, but I am certain that it is a most important adjunct in the treatment of such cases and that the results are very much better in consequence.[2] True, the conditions are not strictly comparable, for in corrosive injuries of the œsophagus a considerable length of the tube is traumatized. But in most cases strips of the mucous membrane escape injury and I have the greatest respect for the natural reparative powers of mucous-membrane-lined tubes. At all events I believe that an early resort to short-circuiting by gastrostomy and cessation of mouth feeding for some weeks is worthy of trial. It seems highly improbable that the results would be worse than by the present method, which is really to leave the result to nature without the assistance that rest might provide. At a later stage the contrast between the clinical behaviour of the œsophagus in swallowing food and its behaviour when under examination by X rays is very remarkable. This I think supports the view, to which experience makes me subscribe, that the act of swallowing largely depends on the pharyngeal muscles, and that the wall of the œsophagus has little power to propel its contents. Even at the present time when the patient is swallowing without difficulty, if the last bites of a meal were followed down the œsophagus they would almost certainly tarry for quite a long time above the narrowing. Nevertheless a column of food with its head subjected to the power of the pharyngeal muscles is readily

propelled through the narrow and probably entirely rigid area. That these conditions are as stated is borne out by the fact that when an œsophagoscopic examination is made in these cases more or less food debris is almost invariably found in the tube above the pathological area even when the condition is under control and the patient is able to swallow satisfactorily. Probably in the case under consideration a length of the œsophagus is converted into a rigid fibrous tube with little or no muscle, or muscle that is rendered functionless by its incorporation in fibrous tissue. The question of the fate of the stenosed area has been dealt with in a previous communication.[1] It is perfectly clear that for a very long time, almost certainly to be measured in years, there will be a tendency to recurrence which must be guarded against in two ways: (1) By the regular use of the bougie; (2) By persistence in the daily use of solid food. Any slight indication of recurrence must not be masked by a resort to food of thinner and thinner consistence. Life can be supported on an entirely fluid diet, but far beyond its monotony should be the knowledge that to accept this limitation is leading up to the development of a vicious circle.

The case also illustrates that no one method can be relied upon in dealing with œsophageal stricture, and that the surgeon must have command of several alternative plans. Of course, other methods might have prevailed, such as the von Hacker retrograde dilatation through the gastrostomy either by bougies or a string of graduated beads. But that method requires the willing and docile co-operation of the patient, and, unfortunately, we had to deal with almost a hostile attitude on the part of our patient, at least for the greater part of the time.

In the difficult management of this case I am much indebted to those of my associates who have already been mentioned, also to Sister Seale, and to Mr. Edgar Herten-Greaven, who was one of the House Surgeons concerned, and who intended to write up the case for publication had it not been necessary for him to return to the Argentine.

Case 2.—**Simple stenosis of the cervical œsophagus following impaction of foreign body, successfully treated by plastic operation.**

The patient was a young man, aged 20, who was first admitted to Hammersmith Hospital on July 27, 1939. He then gave a history that while undergoing sentence he was put to sew sacks, and was using a tailor's thimble with an open end. Without any ulterior motive he put the thimble in his mouth and it accidently slipped over the back of the tongue, and he was unable to retrieve it. This happened on June 22, 1938. From the first he felt some pain in the neck and had some little difficulty in swallowing, which he tried to overcome by mashing up his food. When he complained he was told that the thimble had probably safely passed on downwards. Eventually he was sent to a hospital where the thimble was located in the œsophagus opposite the first dorsal vertebra. An attempt to remove it was not successful, but it was subsequently extracted through the œsophagoscope at another hospital on August 22. He afterwards complained of a cough on attempting to take food, and this prompted a further X-ray investigation which was said to have revealed a tracheo-œsophageal fistula, and on this account a gastrostomy was made. Shortly afterwards the lad absconded, and on recapture was taken to another institution. There he made good progress, and on November 15 a further X-ray examination was carried out, when it was reported that no fistula could be seen, and that the œsophagus was patent in all areas and regular in outline. The patient was regarded as an air-gulper who regurgitated his food. In consequence of this the gastrostomy was closed on November 16, but the patient subsequently informed me that swallowing was not normal at that time and was gradually getting worse. A year later, in June, 1939, the lad again came under medical observation when he stated that he found progressively greater difficulty in swallowing and could then manage fluids only. He had also apparently lost weight.

Further X-ray examination showed that compared with the previous film there was a very marked degree of œsophageal stenosis at the level of the second dorsal vertebra, with marked dilatation of the proximal œsophagus. The X-ray appearances were those of a benign stenosis. As previously, there was much air swallowing. On July 12, a No. 14 œsophageal bougie was passed through an œsophagoscope, and was tightly gripped when in the stricture. A further attempt was made to dilate three days later, but this was unsuccessful. In reporting the details of the history the medical officer wrote: "It would seem probable that the attempted dilatation has provoked œsophageal spasm, as his ability to swallow has been much diminished since."

On admission to Hammersmith Hospital on July 27, 1939, the patient first came under my observation. He was found to be a healthy looking lad of an engaging disposition and in very good general condition and without any external evidence of disability. He could only swallow liquids, and with considerable difficulty. A radiograph sent with the patient showed a gross obstruction opposite the first dorsal vertebra (*Fig.* 174). On August 4 an œsophagoscopic examination by Mr. Sidney Scott disclosed a stricture 9 in. from the teeth. The opening appeared to be eccentric and no bougie could be introduced. After this, attempts were made to get the patient to swallow bougies and a silk thread, but though he made determined efforts, they were unsuccessful. On August 11 it was noted that he had gained 3 lb. in weight, but this was probably due to the attention received from the diet kitchen. On August 17 Mr. Ivor Griffiths examined with the œsophagoscope and demonstrated a considerable pouch with a cicatrix posteriorly with what looked like a lumen in the middle of it, but he was not able to pass a bougie. On August 26 the patient had to be transferred to another hospital on account of the War Emergency, and there his condition got gradually worse and he was altogether very miserable.

FIG. 174.—*Case* 2. The dilated œsophagus above the stricture before treatment at Hammersmith Hospital.

On November 22 the lad was re-admitted to Hammersmith Hospital under my care. He still looked in quite good condition, though obviously thinner, and stated that he was only able to swallow fluids with increasing difficulty. "When he takes some liquid he tries to swallow, after a time regurgitates and swallows again. He has much flatulence, but is unable to belch and cannot vomit. There is no difficulty in breathing, but his friends notice that his voice is thicker than usual. He is progressively losing weight and turns the scale at 10 st. 2½ lb., whereas on his previous admission he was 10 st. 7 lb." No sort of bougie could be passed and he could not swallow a linen thread—in fact the latter seemed to block the passage and made matters worse.

On December 7 gastrostomy was carried out as a preliminary to further treatment, but as his general condition seemed to permit an attempt at retrograde dilatation this was made at the same time. To my surprise I failed to get a bougie to engage in the stricture and the attempt had to be given up. The patient picked up well after the gastrostomy and was buoyed up in the expectation of improvement and gained some weight. In the hope of obtaining the maximum improvement in the local condition by physiological rest, he was only allowed sips of fluid by the mouth and that only with the object of promoting psychological calm. On January 3, 1940, Mr. Ivor Griffiths again made an œsophagoscopic examination but the entrance of the stricture could not be found, and the smallest bougie and even a ureteric catheter failed to pass. During the examination air was introduced through the gastrostomy with a Higginson's syringe, but though the stomach became uncomfortably distended not even a bubble found its way through the stricture. Despite this discouragement I felt that as the patient could still swallow liquid, albeit with great difficulty, this fact at least demonstrated that there was a road through the scar tissue and suggested that a fine bougie

passed by the mouth and encouraged by attempts at swallowing ought to find its way through the narrowing. The lad certainly made determined efforts and on January 8 supposed that he passed a No. 6 followed by a No. 7 bougie. I was present on that occasion and felt satisfied that he had been successful. After this he "passed" larger sizes, but stated that in spite of this apparent success he was swallowing worse than before. On January 15 X-ray examination demonstrated that, alas, the bougies were coiling up in the dilated œsophagus. Further attempts were then made to get the boy to swallow a silk thread, but this just came back into the mouth and the effort was unsuccessful.

By this time the lad was becoming despondent and I felt that we must adopt more radical measures to relieve him. At the last œsophagoscopic examination I was impressed with the difficulty of controlling such a long instrument as a bougie must be if it is to be used through an œsophagoscope. The stricture in this case was situated 9 in. from the teeth and that is a long distance for the control of the effective end of a fine bougie. With these considerations in mind I determined to open the œsophagus above the stenosis as in œsophagotomy, with the idea that this would bring the upper aspect of the stricture within reach and would make its localization and dilatation a much easier proposition. With this object in view, operation was carried out on January 23. Under general intratracheal anæsthesia administered by Dr. Woodfield Davies, a long incision was made down the anterior border of the left sterno-mastoid and the œsophagus was easily exposed on the inner side of the carotid sheath. It looked normal in every way, as were the parts about it. A couple of guide sutures were introduced and the œsophagus incised between them. When opened I was struck by the fact that the wall was neither hypertrophied nor the lumen dilated. On introducing the fore-finger downwards no obstruction was encountered and a bougie of moderate size could be passed to the stomach. In the upward direction an obstruction was reached almost at once which would place it just about an inch below the termination of the pharynx. It was thus apparent that the œsophagus had been opened below the stricture. In view of the appearance of the radiograph (*Fig.* 174) and the distance of the obstruction as measured from the teeth this came as a surprise, but the patient was nearly 6 ft. in height and probably the radiograph taken with the patient standing showed the dilated œsophagus sagged down to some extent. But of course the position of the stenosis was all in favour of the patient, for it made direct plastic intervention possible. An attempt was made to negotiate the stenosis from below, first with different sorts and sizes of small bougies and then with a probe, but this was not successful. A bougie passed from the mouth could be felt by the finger below, but with the œsophageal wall intervening, and no road could be found through the stenosis by that route. The exposure was then extended upwards until the lateral wall of the pharynx was seen above the narrowed area, which appeared as a small mass of fibrous tissue representing a tight annular stricture and without much peri-œsophageal involvement. The œsophagus and pharynx were considerably dilated, but with surprisingly thin walls. The lumen was opened in the same line as the incision below and a further search made, but without discovering any passage through the stenosis. The two incisions were then joined by cutting from the outside through the small area of very tough fibrotic tissue and then only did we expose a narrow mucous-lined channel connecting the œsophagus above and below. The state of the parts disclosed then appeared as shown in the rough diagram (*Fig.* 175). It was intended to make a repair after the fashion of pyloroplasty, but in the event it was really more like an end-to-end anastomosis except that the first stage of the union was represented by the existing channel which had finally been disclosed. The pharynx and œsophagus were easily mobilized and were quite lax, so that

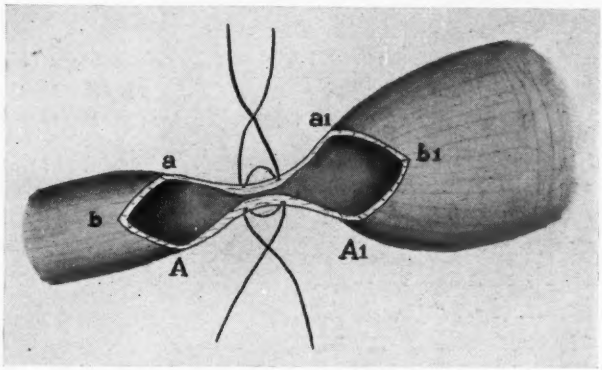

FIG. 175.—Diagram of plastic operation. A sutured to A_1, a to a_1, b to b_1.

there was no difficulty in bringing the parts together. This was done by using interrupted sutures of No. 3/0 chromicized catgut. These were passed without perforating the mucous membrane and the little difficulty in the disproportionate size of the ends was overcome by placing the sutures rather farther apart on the upper end than on the lower. The sutures were tied as they were inserted, but it became clear that the deeper sutures were going to be hidden as the anastomosis proceeded. For that reason a few additional supporting stitches were inserted in the outer coats after every two or three first-layer sutures had been tied. When all had been completed the first layer of sutures was everywhere hidden by these outer sutures, but I question very much if at the conclusion the anastomosis was water-tight. There was very little bleeding and no vessels were tied in the œsophageal wall. While the operation proceeded the cellular tissue by the side of the lower end of the œsophagus was packed with gauze to shut off the path to the mediastinum. At the conclusion of the anastomosis and when the last sutures were cut the re-constructed tube receded into its normal position. The pack in the cellular tissue at the lower end of the incision was replaced by a strand of Bipped gauze, which was brought out through the middle of the incision. A strand of corrugated rubber tissue was brought from the neighbourhood of the suture line and out of the neck just above the gauze. The sternomastoid was allowed to fall back into position, and skin, subcutaneous tissue, and fascia of the upper and lower thirds of the six-inch incision were drawn together by silkworm stitches, the middle of the wound being left open to accommodate the drains. A voluminous dressing was then applied to act as a support for the neck.

FIG. 176.—*Case* 2. Shows incision employed, four weeks after operation.

In order to diminish the risk of infection spreading towards the mediastinum, it had been intended to nurse the patient in the Trendelenburg position, but this was inadvertently omitted until the second day, when the temperature rose to 102°. Despite this rise the patient made no complaint and the pulse was falling. The foot end of the bed was raised, but by the fourth day both temperature and pulse were normal and the patient was again allowed to lie flat. Feeding was entirely by the gastrostomy, and he was directed to eject saliva as far as possible. As a matter of fact recovery was entirely uneventful and I believe that almost from the first he swallowed saliva and without difficulty. The gauze was removed on the sixth day and the rubber drain on the eighth, after which the wound rapidly closed. There was never any sign of leakage and no suggestion of spreading infection. After the operation it was intended to prohibit mouth feeding for two weeks, but we discovered that on the tenth day he surreptitiously ate some chocolate which went down satisfactorily. Three days later feeding per os was "officially" started, and with such success that within a week he was taking practically ordinary diet. This went down quite easily, but he stated that he felt it passing over a rather sore area. Further radiographs showed a considerable degree of patency, a medium as thick as porridge going down without difficulty. There was still some dilatation in the proximal part of the gullet. He then weighed 9 st. 4 lb. On February 24 the X-ray report was practically the same, but by this time the boy was taking his ordinary food with the rest of the patients in the ward, and was obviously gaining weight and the wound was all but healed (*Fig.* 176). As a precaution a Hurst's bougie was passed, the largest size slipping down with ease.

On March 8* he was discharged from hospital. At that time he was able to take ordinary food without difficulty and weighed 11 st., a gain of 24 lb. since the operation. He was very cheerful and was looking forward to returning to the North to take up work in a munition factory.

* In July, 1940, he reported and was quite well.

Comment.—In my limited experience the impaction of a foreign body is a rare cause of stenosis. In the collection of foreign bodies in the œsophagus preserved in the Museum of the Medical School at Newcastle-upon-Tyne there is no example of such a condition. Nevertheless, it is well recognized as among the possible causes, and this case is a further reminder of the necessity for the early recognition and early removal of foreign bodies. The next point is the very limited nature of the lesion, for it astonished me to find so small a mass of fibrous tissue and so short a length of œsophagus involved. Dilatation above the stricture, on the other hand, was much more than one is accustomed to see in such a comparatively short time, but that was probably partly accounted for by the high situation of the lesion and the distensibility of the pharynx and upper œsophagus. The difficulty, or I should rather say, my difficulty, in the exact localization of the situation of the obstruction was interesting. It certainly emphasizes the importance of taking the height of the individual and especially the length of the neck into consideration, at least when taking measurements from the teeth. Nevertheless, the situation was extremely fortunate from the point of view of the patient, and indeed it is much to be desired that other non-malignant strictures might be so fortunately situated. Plastic operation was comparatively easy to carry out, and the immediate result was most satisfactory, both to the patient and the surgeon. I should have preferred to defer the record of the case until a lapse of twelve months or more, but the uncertainty of the times in which we live colours one's outlook in these matters and has prompted this present communication. But I feel that the result will probably continue to be satisfactory. I have the greatest respect for the power of the pharyngeal muscles as dilators, for they are capable of forcing a bolus of food through a very marked constriction, and especially is that the case when the narrowing is so near to the pharyngeal muscles themselves. I have elsewhere[1] recorded a case where a very troublesome stricture at the commencement of the œsophagus was treated by self-dilatation with bougies and for five years the patient has not used a bougie and there has been no symptom suggesting recurrence. The principle of pyloroplasty has always appealed to me, and I can never forget the really brilliant results which I used to see years ago when the operation was employed in the non-malignant stenoses of the pylorus.[3] It is a wonderful means of dealing with simple strictures when the lumen of the tube concerned and the nature of its walls lend themselves to its employment. But of course it is only suitable for narrowings of limited extent and, unfortunately, only those at the extremities of the œsophagus are accessible. In the thoracic œsophagus fibrous strictures often involve a considerable length of the wall, and this has to be borne in mind as well as the difficulties associated with their exposure.

For much help in the management of this case I must thank my assistant, Mr. A. Lawson Light, my House Surgeon, Mr. J. H. A. Jewell, and, not least, Sister Mathias.

I am also indebted to Dr. Alan Daley, the chief Medical Officer of the London County Council, for permission to report these cases.

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LEIOMYOMATA OF THE STOMACH

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Our interest in this subject was aroused by two recent cases, one of which (*Case* 1) presented a very characteristic picture at gastroscopy. We had also remembered a third case successfully operated upon by Mr. Arthur Edmunds some years ago, and the three provide a small series which seems worthy of recording.

CASE REPORTS

Case 1.—O. M., a male, aged 70, was referred to King's College Hospital by Dr. P. H. Taylor, in January, 1938. He had complained of pain after meals for the past eighteen months. There were no marked periods of remission of pain. He had suffered from nausea and flatulence. Six weeks prior to admission he had had diarrhœa, passing black motions, and for the last few days there had been epigastric pain.

On Examination.—Except for some pallor of the mucous membrane, nothing abnormal was found.

Blood-count.—

R.B.C.	5,280,000	Hæmoglobin	52 per cent
W.B.C.	9,000	Colour Index	0·79

Gastric Residuum.—Total acid, 0·040 g. per cent; no free acid; blood and mucus present.

Fig. 177.—*Case* 1. X-ray photograph showing outline of rounded tumour at the fundus.

Fig. 178.—*Case* 1. Specimen removed at operation. The surface of the tumour has ulcerated.

X-ray Report (Dr. A. H. Rackow).—" Examination of the stomach yielded an unusual appearance. An oval clear area is seen to persist in the upper part of the body of the stomach. In all views the outline of the stomach is normal, which would exclude any extensive attachment of this filling defect to the walls of the stomach. The finding is less pronounced when the stomach is filled than in the earlier stages of filling. Evidence of the presence of a large rounded radiotranslucent body in the stomach is very strong and suggests one of several possibilities, fibroma on a pedicle, a mass of mucus, or foreign material. There is no evidence of an ulcer in the stomach or duodenum." (*Fig.* 177.)

Gastroscopy.—There is a large polypoid mushroom-like mass in the fundus of the stomach. It is ulcerated on its surface. The gastric mucosa is of atrophic type. The tumour is not necessarily malignant and is possibly an ulcerated simple tumour (? leiomyoma).

OPERATION (January 2, 1939).—Left paramedian incision; examination of the stomach suggested the presence of a pedunculated tumour attached just below the gastro-œsophageal junction and near the greater curvature. A sleeve resection of the part of the stomach containing the growth (*Fig.* 178) was performed and the abdomen closed with drainage.

PROGRESS.—Convalescence was protracted owing to the development of a pleural effusion. The patient was finally discharged from hospital eight weeks after the date of his operation.

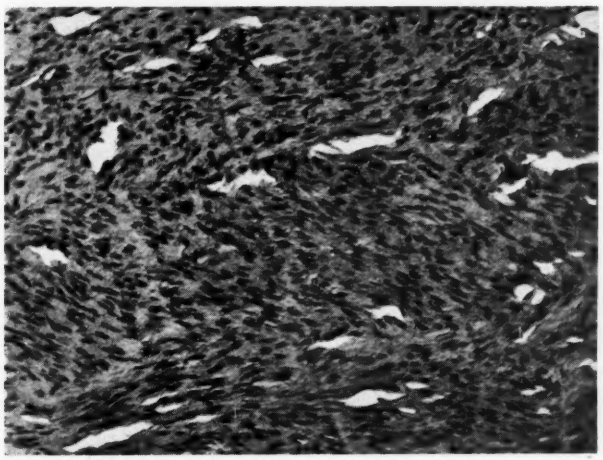

FIG. 179.—*Case* 1. Microphotograph of a portion of the tumour. (× 200.)

HISTOLOGY (Dr. E. ff. Creed).—" The tumour is a leiomyoma and it is situated in the submucosa. It is very cellular and it is composed of interlacing bundles of unstriped muscle. Occasional mitoses can be seen. It is surrounded by a capsule and is not invading it. There is no evidence of malignancy." (*Fig.* 179.)

Case 2.—W. I., a male, aged 68, was referred to King's College Hospital by Dr. D. McCarthy, in October, 1938, complaining of upper abdominal pain and flatulence for the past two years. The pain lasted for a few minutes and was fairly severe while it lasted. It bore no relation to food and was not relieved by taking food. The appetite had been good, but he said he had lost a good deal of weight lately.

ON EXAMINATION.—Nothing abnormal was discovered on abdominal examination.

Gastric Residuum.—Before histamine, total acid 0·033 g. per cent; no free acid. After histamine, total acid 0·288 g. per cent; free acid 0·234 g. per cent. Blood was present in both specimens.

X-ray Report (Dr. G. Doel).—" A large filling defect on the greater curvature of the stomach just below the fundus. The mucosa in this region was freely mobile. The condition appeared to be one of neoplasm of the greater curvature." (*Fig.* 180.)

Gastroscopy.—The distal half of the stomach appears to be normal. The proximal half shows well-advanced atrophic gastritis. Projecting from the posterior wall about 2 in. from the lac muqueux is a non-ulcerated mass which appears to be a growth of the stomach

itself. Proximal to this projection is a pouch. It is possible that the appearances might be due to an extragastric tumour pushing the stomach wall inwards into the lumen.

OPERATION.—The stomach was examined, but no evidence of a carcinoma could be found. Exploration of the lesser sac revealed the presence of a subserous tumour the size

FIG. 180.—*Case* 2. Note the deformity of the greater curvature, which persisted in all the films taken.

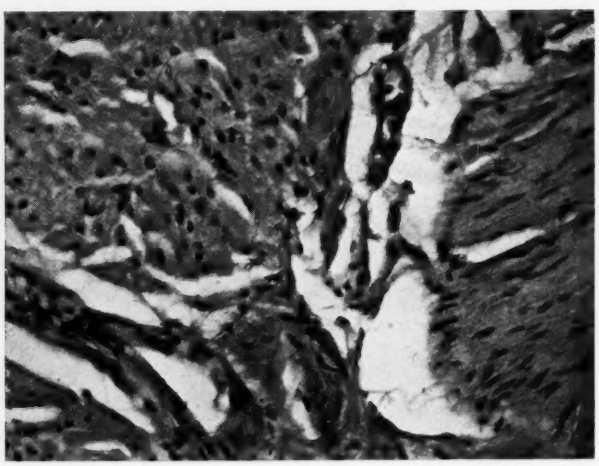

FIG. 181.—*Case* 2. Microphotograph of a section of the tumour. (× 200.)

of a hazel-nut and situated in relation to the posterior wall of the stomach at about the area of deformity noted on X-ray examination and at gastroscopy. The tumour was removed, together with the underlying portion of the stomach wall. The opening into the stomach was closed and the peritoneum and wound sutured in the usual layers.

Convalescence was uneventful.

HISTOLOGY (Dr. E. ff. Creed).—" The tumour is a leiomyoma occurring between the muscular and serous coats of the stomach. It is composed of interlacing strands of large spindle-shaped cells having a somewhat whorled arrangement with a little intervening fibrous tissue. The cells have elongated pale-staining nuclei and fibrillary cytoplasm, which appears somewhat degenerate; they resemble normal smooth muscle-cells; where the tumour abuts on the normal muscle tissue the edge is rather indefinite. There is no evidence of malignancy." (*Fig.* 181.)

Case 3.—A. C., a female, aged 49, was admitted to King's College Hospital in August, 1929, under the care of Sir Charles Briscoe and Mr. Arthur Edmunds on account of profound anæmia. She had collapsed in the street, and subsequently had passed a number of fluid, tarry motions. She had suffered from severe indigestion for two to three years prior to admission.

ON EXAMINATION.—Apart from pallor due to anæmia, nothing abnormal was discovered.

Blood-count.—

R.B.C.	3,175,000	Hæmoglobin	30 per cent
W.B.C.	4,200	Colour Index	0·47

Gastric Residuum.—Total acid 0·117 g. per cent; no free acid; blood and mucus present.

X-ray Report (Dr. Graham Hodgson).—" There is an abnormal, translucent area in the stomach near the fundus. Screening failed to fill this area even when pressure was used." (*Fig.* 182.)

FIG. 182.—*Case* 3. Translucent area due to the presence of polypoid growth.

FIG. 183.—*Case* 3. Gastrotomy and removal of growth, which could be moved freely over the muscularis. (*Drawn after operation by Mr. Arthur Edmunds.*)

OPERATION (Mr. Arthur Edmunds).—A tumour could be felt inside the cavity of the stomach. An incision through the anterior wall of the stomach was made and a polypoid growth, 3½ in. by 2½ in. was found growing from the posterior wall (*Fig.* 183). It moved freely over the muscularis and was easily removed by an incision through the mucosa encircling pedicle. The operation specimen is illustrated in *Fig.* 184.

Convalescence was satisfactory.

HISTOLOGY (Dr. E. ff. Creed).—" The tumour is a leiomyoma. It consists of a very cellular fibrous tissue with interlacing bundles of unstriped muscle-cells. The fibrous tissue is extremely vascular, the vessels having a well-defined endothelial lining. The growth is

encapsulated and at one area the capsule is broken, the rent showing continuity with the lumen of an artery in the periphery of the tumour."

The patient was re-admitted in March, 1937, under the care of Mr. C. P. G. Wakely, suffering from an inoperable carcinoma of the cæcum, with secondary deposits in the omentum.

FIG. 184.—*Case* 3. Photograph of operation specimen. The bristle indicates the site of an ulcer which has eroded a blood-vessel.

COMMENTARY

Leiomyoma appears to be the commonest simple tumour of the stomach. Eliason and Wright estimate that 60 per cent of all simple gastric tumours are leiomyomata, and Easterman and Sentry found that of 27 cases of simple growth, 10 were leiomyomata. Minnes and Geschickter quote the following relative incidence of simple tumours arising from mesenchyme in 522 cases from the literature :—

1. Leiomyomata	341 (65·3 per cent)
2. Neurofibromata	102 (19·5 per cent)
3. Fibromata	42 (8·0 per cent)
4. Lipomata	32 (6·1 per cent)
5. Osteomata	1 (0·19 per cent)
6. Osteochondromata	1 (0·19 per cent)
7. Myomata	3 (0·57 per cent)

Conway was able to trace records of 58 cases of leiomyomata, 2 of which occurred in his own practice. In 33 the tumour was discovered post mortem, and, of these, 13 had died as a result of some other unassociated disease.

There does not appear to be any particular age or sex incidence, the condition being evenly distributed in both sexes, and the variation in age incidence is shown by the fact that the youngest recorded was in a child of 9 years, and the oldest in a man of 78.

The tumour may vary in size from that of a pea to that of a man's head. It may be subserous, intramural, or submucous in position. Most of those giving rise to symptoms are of the submucous variety. As Conway found,

leiomyomata are not infrequently found at post-mortem examination, having given rise to no symptoms during life.

The leiomyoma is encapsulated, but the mucosa covering it may ulcerate. According to Starr Judd, a true peptic ulcer is sometimes found in the mucosa overlying the growth, but it is more usual for the ulceration to be due to interference with the vascularity of the mucosa by the growth itself.

The tumour is liable to undergo malignant degeneration and produce secondary deposits, particularly in the liver; Patterson collected 14 such cases. An interesting feature in this respect is that the tumour itself may show little or no evidence of malignant change yet the secondary growths present the picture of leiomyosarcomata. Stewart, of Leeds, has compared their tendency towards malignancy with that of uterine fibroids.

Leiomyomata may give rise to symptoms indistinguishable from those of carcinoma of the stomach (Lindguist and Mock; Swan and Cooper). Loss of weight and of appetite are probably due to associated atrophic gastritis, a condition which was demonstrated by gastroscopy in *Cases* 1 and 2. Epigastric discomfort may be caused, according to Conway, by interference by the growth of the normal mobility of the stomach wall.

Severe hæmorrhage, due to ulceration of the surface of the growth, may lead to a profound secondary anæmia, and was the outstanding symptom in the first of our cases. Conway, in his analysis of 33 untreated cases found at post-mortem examination, states that 6 died as a result of massive gastric hæmorrhage. If the tumour is pedunculated and growing from the region of the pyloric antrum, it may be carried into the pyloric canal and cause pyloric obstruction—a ball-valve obstruction—or alternatively produce partial obstruction by reason of its size alone.

Diagnosis.—Leiomyoma is curable by operation although it may have reduced the patient to a state comparable with that caused by an extensive malignant growth. Hence its recognition and differentiation from carcinoma is of very great importance. An exact diagnosis, we feel, is only possible when the leiomyoma is submucous in position and forms a pedunculated tumour projecting into the cavity of the stomach.

Pansdorf and Determann report 4 cases correctly diagnosed before operation by X-ray investigation. Moore points out that if the following radiological features are not characteristic, they are at least suggestive :—

1. A filling defect that is circumscribed and punched-out in appearance.
2. The filling defect is usually on the gastric walls, leaving the curvatures regular and pliant.
3. While the rugæ are obliterated in the immediate area of the tumour, the rugæ surrounding the tumour are normal in their arrangement and distribution.
4. They cause little or no disturbance in peristalsis and retention is uncommon except when the tumour is situated near the pylorus.
5. They do not reveal a niche, nor is there any evidence of an incisura or spasm.

Gastroscopy will readily give confirmation. The tumour is easily seen, is pedunculated, and though atrophic gastritis may be present, the base of the tumour is sharply defined at its attachment to the stomach wall.

Treatment.—The treatment is operative, the scope of operation ranging from that of a partial gastrectomy to that of a gastrotomy and local removal of the tumour. All writers are agreed as to this even if the tumour is symptomless, because of the risk of hæmorrhage and the possibility of malignant transformation.

Case 3 illustrates well that the cure may be achieved by removal of the growth and an encircling area of mucosa only, always provided that the pedicle is free from the muscular coat. If the pedicle is adherent, local removal of the whole thickness of the stomach wall at the attachment of the pedicle will suffice. No extensive form of gastrectomy is necessary or desirable. On reflection after operation in *Case* 1, it was felt that the amount of stomach removed was far in excess of requirement, though, happily, without harm.

We were unable to find any definite record of the operative mortality for this condition, but Judd and Hoerner, discussing the operative mortality of 50 cases of all varieties of simple tumours of the stomach, record 1 death. The remainder derived lasting benefit. All three cases we have recorded made satisfactory recoveries.

SUMMARY

Three cases of simple tumour of the stomach are recorded; all proved to be leiomyomata.

Symptoms may be caused by ulceration, or obstruction at the pylorus, and may closely simulate those of carcinoma.

Gastroscopy is an important aid to diagnosis.

A brief résumé of the literature is given.

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THE SPREAD OF ACUTE INTRAPERITONEAL EFFUSIONS

By G. A. G. MITCHELL, Aberdeen

Fluids escaping within the peritoneal cavity from an acute visceral perforation are guided by a series of natural barriers towards certain potential spaces where they tend to collect. Various observers have described these barriers and spaces and their influence on the spread of effusions, but a critical examination of their descriptions and diagrams reveals errors and discrepancies. Accordingly the following experimental, anatomical, and clinical evidence is submitted to show that the spread of intraperitoneal fluids is not completely in accordance with the usual descriptions.

According to accepted teaching, material escaping from a perforated duodenal ulcer passes down to the ileocæcal region, along the right external paracolic sulcus, but in the careful investigation of a number of these cases coming to early operation, several showed recognizable stomach contents in the ileocæcal region, while the upper part of the right external paracolic sulcus was apparently uncontaminated; again, in standard descriptions, the stomach contents flow from the right external paracolic gutter into the pelvis, and then into the left infracolic space, but it is found in practice that the left infracolic space is sometimes contaminated when the pelvis remains unaffected; yet again, the standard description that fluid from a perforated duodenal ulcer readily escapes from Morison's pouch into the right subphrenic space at an early period is refuted by the fact that shoulder pain is not a characteristic early symptom in this condition.

Before discussing the experiments whereby the spread of an effused fluid from a perforated viscus was studied under as natural conditions as possible, it is necessary to describe the various barriers and spaces, because the text-book descriptions not only vary, but are often confusing, while many are inaccurate in anatomical details. The following description is based in part on the writings of Morison, Barnard, Box, Treves, Livingston, and Lee McGregor, but mainly on the experimental results to be cited, and on personal observations in the operating theatre and dissecting room.

ANATOMICAL CONSIDERATIONS

1. THE INTRAPERITONEAL BARRIERS OR WATERSHEDS

The spread of an effusion within the peritoneal cavity is governed by the same physical laws which regulate the flow of fluids elsewhere. Thus it runs down slopes, accumulates in depressions, and tends to be retained in them by certain natural barriers; and although the spread may be influenced by such factors as the position of the patient, the site of perforation, the nature of the escaping visceral contents, the rapidity of outflow, the state of distension of the hollow viscera, the presence or absence of adhesions, and anatomical variations in the size and disposition of the abdominal contents, it is found in practice that the paths followed by the effused fluid, and the spaces where it collects, are similar in nearly every case of acute perforation of the same viscus.

In the recumbent position the lumbar portion of the vertebral column and the posterior part of the pelvic brim project considerably above the level of the adjacent parts of the abdominal parietes, and along with their overlying structures, they present the appearance of ridges separating three hollows or depressions, two paravertebral and one pelvic; the paravertebral depressions are in turn divided into upper and lower parts by the transverse colon and mesocolon. These natural ridges and hollows have been compared to geological watersheds and catchment basins, a picturesque conception which facilitates the understanding of the spread of intraperitoneal fluids. But the comparison cannot be pushed too far, for as the intraperitoneal watersheds are partly composed of soft tissues, they are liable to variations and displacement, and the catchment basins which exist in the supine position may disappear entirely when the patient assumes the erect position. Besides, there are many secondary barriers in the shape of viscera, peritoneal folds, adhesions, etc., which may arrest or divert the streams descending from the watersheds, and their importance in influencing the spread of peritoneal effusions is never sufficiently stressed.

It is customary to describe three great barriers or divides, but it must be realized that their action as watersheds is pronounced only when the body is in the supine position. This fact, however, does not invalidate deductions based on their existence, because the patient with an acute perforation almost invariably adopts the recumbent attitude.

1. The Longitudinal Barrier.—The bodies of the lumbar vertebræ, the aorta, the inferior vena cava, and the mesentery of the small intestine form a longitudinal barrier, dividing the abdominal cavity, below the level of the transverse colon, into two great regions or spaces—right and left infracolic (*Fig.* 185). As the line of attachment of the mesentery passes from the left side of the body of the second lumbar vertebra above to the right sacro-iliac joint below, the general direction of this barrier is slightly oblique, and thus the right and left infracolic spaces are not symmetrical.

2. The Lower Transverse Barrier.—The posterior part of the pelvic brim, along with the overlying psoas muscles and iliac vessels, projects forwards and tends to separate the abdominal and pelvic cavities. In the supine position the projection caused by these structures lies at a distinctly higher level than the floors of the adjacent hollows, formed by the paravertebral fossæ and the pelvic cavity, and thus fluids effused above the level of the sacral promontory tend to flow upwards towards the diaphragm, while effusions below this level collect in the pelvis.

3. The Upper Transverse Barrier.—The transverse colon and mesocolon form the third great barrier, dividing the abdominal cavity into supracolic and infracolic regions (*Fig.* 185). The root of the transverse mesocolon is attached in front of the duodenum, pancreas, and great vessels, about the level of the upper border of the second lumbar vertebra, but it rises slightly at both ends to the level of the body of the first lumbar vertebra. The left colic flexure is usually at a distinctly higher level than the right colic flexure, but the difference between the levels of attachment of the corresponding ends of the root of the mesocolon is not so great. The line of attachment of the root of the mesocolon is therefore moderately concave upwards, but, taken as a whole, the upper surface of the mesocolon slopes gently downwards from left to right and from behind forwards. The mesocolon itself is narrow or absent on the right side, so that the proximal third of the transverse colon is held

back towards the posterior abdominal wall and has less freedom of movement than the central and distal parts. The relative immobility of this part is well known to anyone who has performed a number of transverse colostomies, and it is not uncommon to find that there is no appreciable mesocolonic width except to the left of the midline, the gut being attached directly to the head of the pancreas and the front of the second part of the duodenum by areolar tissue. This anatomical arrangement has a bearing on the spread of fluids from the right subhepatic compartment.

II. THE INTRAPERITONEAL DRAINAGE BASINS

The three great barriers or watersheds divide the peritoneal cavity into four main potential spaces (*Fig.* 185): (1) Supracolic; (2) Right infracolic; (3) Left infracolic; and (4) Pelvic; and some of these, in turn, are divided up into smaller compartments. Normally none of these spaces is completely isolated, though it may become so as the result of pathological processes.

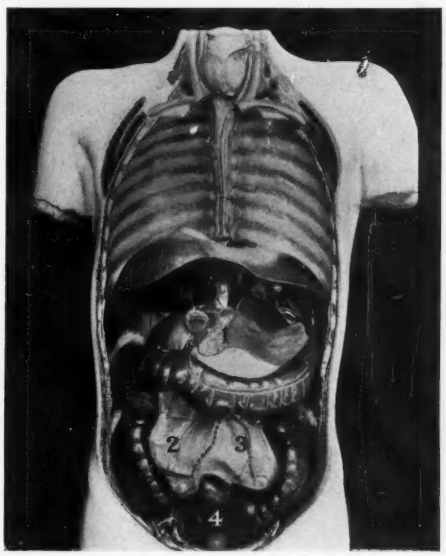


FIG. 185.—The intraperitoneal drainage basins. The liver, stomach, and small intestines have been removed. The dotted line represents the attachment of the mesentery. 1, The supracolic space; 2, The right infracolic space; 3, The left infracolic space; 4, The pelvic space.
(*Photograph of His's model.*)

1. The Supracolic Space.—This lies below the diaphragm, and above the transverse colon and mesocolon. It contains such important structures as the liver, gall-bladder, stomach, first part of the duodenum, and spleen, and lies in front of the pancreas, kidneys, suprarenals, and great vessels. It communicates in front of the transverse colon and greater omentum, and along the sides of the latter structure, with the infracolic spaces. If the greater omentum is voluminous it may envelop the small intestines and the ascending and descending colons, both anteriorly and laterally, and hang down into the pelvis. This type of omentum is uncommon, particularly in the adult.

The supracolic space is broken up, mainly by the liver and its ligaments, into smaller compartments, so making this the most complicated of the potential intraperitoneal spaces. A varying number of these are described (4–6), but most writers now base their classification on Barnard's description—namely, that there are right and left anterior and right and left posterior intraperitoneal spaces in the supracolic region of the abdomen. This terminology, though it has the authority conferred by fairly long usage, is misleading and should be discarded, or, if it is retained, the spaces should be called right and left antero-superior and right and left postero-inferior intraperitoneal spaces. These clumsy terms can be avoided by referring to the spaces lying immediately beneath the diaphragm as subphrenic, and those lying mainly beneath the liver as subhepatic. Four spaces may then be simply defined in the supracolic region: *right and left subphrenic and right and left subhepatic*, all extensive and irregular in outline.

a. The Right Subphrenic Space.—This is situated between the diaphragm and the superior, right, and anterior surfaces of the right lobe of the liver (*Fig.* 186), and separated from the corresponding space on the left side by the falciform ligament (*Fig.* 187). Posteriorly it is limited by the superior layer of the coronary ligament which separates it from the right posterior extraperitoneal space. Below it opens towards the right infracolic space, in front of the transverse colon and greater omentum, and communicates round the sharp inferior border of the liver with the right subhepatic space (*Fig.* 186), while on the right side it is continuous with the right external paracolic groove. The experiments described later show that the freest pathway of communication with the right subhepatic space is through the quadrangular interval bounded by the inferior edge of the quadrate lobe of the liver above, the transverse colon below, the fundus of the gall-bladder on the right side, and the free edge of the falciform ligament on the left side. The right and left subphrenic spaces communicate through a space bounded above and in front by the free margin of the falciform ligament, below by the upper surface of the transverse colon, and behind by the pyloric end of the stomach. It is evident, therefore, that the region below and on either side of the free margin of the falciform ligament is a sort of junction where the two subphrenic and the right subhepatic spaces communicate; and a diverticulum of the left subphrenic space, sometimes termed the left anterior subhepatic space, also communicates with the right subhepatic space in this region.

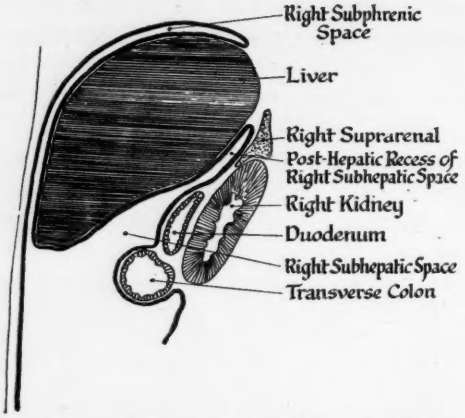


FIG. 186.—A sagittal section through the abdomen to the right of the median plane. Diagrammatic.

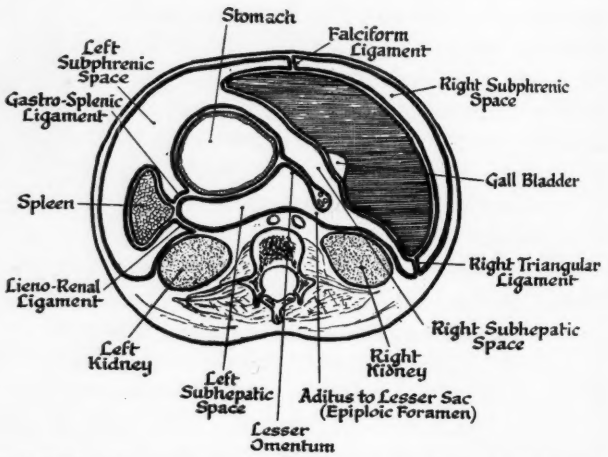


FIG. 187.—A horizontal section through the upper part of the abdomen. Diagrammatic.

b. The Left Subphrenic Space.—This is the potential cavity lying beneath the diaphragm, between it and the superior and anterior surfaces of the left lobe of the liver, the antero-superior surface of the stomach, and the diaphragmatic surface of the spleen (*Figs.* 187, 188). Anteriorly, in the quadrangle formed by the left costal margin, the inferior border of the liver, the falciform ligament, and the transverse colon, it is bounded also by a small area of the anterior abdominal wall. It is separated from the right subphrenic space by the

falciform ligament, but communicates with this space through the interval between the falciform ligament, transverse colon, and stomach. Posteriorly, it reaches as far as the superior layer of the left triangular ligament, and postero-internally it is separated from the lesser sac (left subhepatic compartment) by the lienorenal ligament (*Fig.* 187). Below it opens towards the left infracolic space in front of the transverse colon, left colic flexure, and greater omentum, and may communicate with the left external paracolic groove, but usually there is a peritoneal fold stretching from the left colic flexure and left upper extremity of the greater omentum to the diaphragm opposite the tenth and eleventh ribs (the phrenicocolic ligament or sustentaculum lienis) which forms a barrier between the space and the external paracolic groove.

The spleen actually projects into this compartment, as can be appreciated from an examination of the diagram (*Fig.* 187), and in certain cases it gives attachment to a peritoneal fold of varying width, the phrenicosplenic ligament, extending from the upper extremity of the spleen to the contiguous part of the diaphragm.

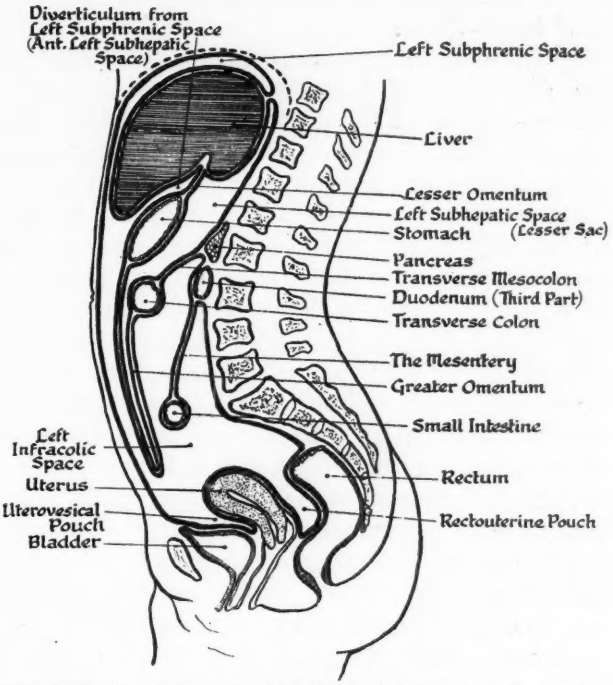


FIG. 188.—A sagittal section through the abdomen to the left of the median plane. Diagrammatic.

The interval between the inferior surface of the left lobe of the liver, the anterior surface of the hepatogastric portion of the lesser omentum, and part of the antero-superior surface of the stomach (*Fig.* 188), may be regarded as a diverticulum extending upwards and backwards from the antero-inferior part of the left subphrenic space, though theoretically it would be more correct to describe it as a subhepatic compartment. Normally it communicates freely with the rest of the left subphrenic space through the interval between the liver and stomach, and with the right subhepatic space through the interval bounded by the ligamentum teres above, the hepatoduodenal ligament behind, and the transverse mesocolon below. Posterolaterally it extends backwards to the spleen and the gastrosplenic ligament. Several writers dealing with the subject of subphrenic abscess describe this interval as the left anterior subhepatic space, in contradistinction to the lesser sac, which is then regarded as the left posterior subhepatic space. There is considerable justification for this from the pathological viewpoint, since in subacute and chronic conditions the recess often becomes isolated by adhesions, and Oschsner and Graves state that it is the second most common site for a subphrenic abscess. But normally there are no such adhesions, the space communicates widely with the main part of the left

subphrenic space, and thus there are no anatomical barriers preventing the spread of an acute effusion from a perforated viscus in this compartment.

c. The Right Subhepatic Space.—Great interest has been evinced in this space since it was first described by Morison in 1894 as " a pouch below the right lobe of the liver and gall-bladder separated from the general peritoneal cavity by natural barriers ". Succeeding writers have named it the right kidney pouch, the right renal well, the right hepatorenal fossa, the right posterior intraperitoneal compartment, the right inferior intraperitoneal space, the right postero-inferior intraperitoneal space, and Morison's pouch. It is doubtful if any of these names is superior to the term ' right subhepatic space ', though to those with historical leanings the eponym is attractive.

The space or pouch (*Figs.* 186, 187) is bounded above and in front by the inferior surface of the right lobe of the liver and by the gall-bladder: below and behind by part of the right kidney, the right suprarenal gland, the second part of the duodenum, the right colic flexure, the transverse mesocolon, a small part of the head of the pancreas, and the crural fibres of the diaphragm. Behind it projects upwards in the form of a recess between the renal impression of the liver in front, and the upper pole of the right kidney, the lower part of the right suprarenal gland, and the right half of the inferior vena cava behind. This recess (*Fig.* 186) is limited above by the inferior layer of the right coronary ligament, and communicates around the margin of the right triangular ligament with the part of the subphrenic space lying between the right surface of the liver and the diaphragm, while below it opens towards the right external paracolic groove. Morison and several later writers describe the external paracolic groove as a part of the right subhepatic space, but as it is largely below the level of the transverse barrier, it is more logical to regard it as a subdivision of the right infracolic space. Moreover, the groove is often almost completely separated from the subhepatic space by a peritoneal fold, known as the hepatorenal ligament, stretching between the inferior surface of the liver, the right colic flexure, and the front of the right kidney. The posthepatic recess of Morison's pouch is the lowest part of the right lumbar paravertebral groove when the body is in the supine position, and so effusions tend to gravitate there. Apparently it becomes readily isolated from the main part of the right subhepatic space by the formation of adhesions, and it is the commonest site of a subphrenic abscess.

On the right side Morison's pouch is bounded more or less completely by the hepatorenal ligament, but when this ligament is not well developed it communicates with the right external paracolic groove through the interval between the liver, kidney, and right colic flexure. On the left side it extends almost to the midline, the boundaries being the inferior vena cava, the caudate lobe of the liver, the first part of the duodenum, and the hepatoduodenal ligament. These structures surround the epiploic foramen, and through this opening the pouch communicates with the lesser sac or left subhepatic space (*Fig.* 187). Below, it communicates with the right infracolic space in front of the proximal part of the transverse colon and the right colic flexure. Anteriorly, it communicates with the antero-internal angle of the right subphrenic space through an interval bounded by the fundus of the gall-bladder, the inferior border of the quadrate lobe of the liver, the free edge of the falciform ligament, and the transverse colon; this communication was mentioned before, and it was emphasized that the right and left subphrenic spaces communicate in this region, below the falciform ligament and above the transverse

colon. Thus an effusion may spread from the right subhepatic pouch into the lowest part of the right subphrenic space, and from there into the corresponding left subphrenic space. The right subhepatic pouch also communicates with the diverticulum from the left subphrenic space which lies between the liver, lesser omentum, and stomach (the left anterior subhepatic space), through a space bounded by the quadrate lobe and ligamentum teres above, the hepatoduodenal ligament behind, and the transverse mesocolon below. Occasionally this pathway of communication is greatly narrowed by the presence of an inconstant peritoneal band, the cystogastrocolic ligament, which stretches from the gall-bladder to the pyloroduodenal junction, or may extend across the latter to become attached to the transverse colon and mesocolon.

d. The Left Subhepatic Space.—This space is really the lesser sac or omental bursa of the peritoneum (*Figs.* 187, 188). Anteriorly it is bounded by the lesser omentum, the postero-inferior surface of the stomach, and the commencement of the duodenum; posteriorly by most of the pancreas and left suprarenal gland, a small part of the anterior surface of the left kidney, the diaphragm, and the upper parts of the abdominal aorta and inferior vena cava; above by the caudate lobe of the liver, which projects into the sac; below by the fusion of the constituent layers of the greater omentum; on the left side by the lienorenal and gastrosplenie ligaments and the splenic hilum; and on the right side it extends just to the right of the midline where it opens into the right subhepatic pouch through the epiploic foramen (aditus to lesser sac).

The lower boundary of this compartment is somewhat variable, depending on the extent of fusion of the layers of the greater omentum, and it is usually stated that this fusion is less extensive in the young, and that a space often exists practically as far as the lower border of the greater omentum. The lower limit of the lesser sac was one of the points specially investigated in the experiments described below. It was found in 31 experiments that it seldom extended much below the level of the transverse colon, and never approached the lower margin of the greater omentum, though in nearly every case the barium spread a little lower down on the left side than on the right. The lowest level reached in any specimen was the lower border of the body of the fourth lumbar vertebra, and the series included several immature fœtuses, but in most cases the lower limit was nearer the level of the lower border of the second lumbar vertebra.

The interior of the lesser sac is partly subdivided by two sickle-shaped peritoneal projections, the right and left gastropancreatic folds. The former is produced by the left gastric artery as it passes from the cœliac axis to the lesser curvature of the stomach, and the latter by the hepatic artery as it passes forwards beneath the epiploic foramen to insinuate itself between the layers of the lesser omentum. The size of the folds varies considerably, but when well marked they tend to subdivide the space into a smaller superior and a larger inferior recess.

This compartment always communicates with the right subhepatic pouch through the epiploic foramen, and it may communicate with the subhepatic diverticulum of the left subphrenic space if the lesser omentum is of the cribriform type. Unless it is directly involved, however, as in perforation of a posterior gastric ulcer, pancreatitis, etc., it often escapes infection in spreading peritonitis, because the epiploic foramen readily becomes sealed, and the space itself may become obliterated by the formation of adhesions.

2. The Right Infracolic Space.—The right infracolic space lies below the proximal half of the transverse colon and mesocolon and to the right side of the mesentery (*Fig.* 185). On the anterior and right sides are the abdominal parietes, separated more or less completely from the space by the folds of the greater omentum. Posteriorly there is the lower part of the right lumbar paravertebral groove or depression, which is partly levelled up by the presence of such structures as the lower pole of the right kidney, the perinephric and paranephric fat, part of the duodenum, the right psoas and quadratus lumborum muscles, and the ascending colon.

Owing to the oblique disposition of the mesenteric root this space is widest above, and ends below in an angle in the region of the ileocæcal junction. It contains coils of small intestine, most of the cæcum, and the ascending colon if it possesses a mesocolon. When the appendix occupies the paracolic position it lies partly in this space, and when it occupies the splenic or ' 2 o'clock ' position its tip may pass in front of the lower ileum and so enter the space, but more often the whole organ lies below the ileum in the left infracolic space.

The ascending colon ascends almost vertically through the space, and along each side there is a gutter or channel, the right external and internal paracolic grooves (*Fig.* 185). The right external groove has achieved notoriety because it is supposed to provide an easy pathway for the spread of infection upwards or downwards between the right subphrenic and subhepatic spaces and the ileocæcal region. While effusions do spread in this way, especially from the ileocæcal region towards the diaphragm, it should not be imagined that the right external groove is a simple smooth channel offering an unimpeded pathway for the free flow of fluids. It is usually crossed by one or more crescentic peritoneal folds, most of which are relatively small, but one or two are larger, and all tend to hinder the flow of fluid. The largest fold is inconstant, and stretches from the flank, about the level of the right inferior margin of the liver, to the outer and anterior surfaces of the ascending colon. At the lower end the cæcal fold, which extends between the iliac fossa and the posterolateral aspect of the cæcum, forms an incomplete barrier between the right external groove and the pelvis. Above, the hepatorenal ligament may partially or completely isolate the right subhepatic space from the right external paracolic groove.

The right internal paracolic groove is shallow, and terminates below at the ileocæcal junction, while above it ends in a blind recess below the right colic flexure. At its lower end it communicates with the termination of the right paramesenteric gutter, which may be defined as the groove between the right side of the root of the mesentery and the posterior abdominal parietes. Infective material occasionally spreads along one or other of these channels.

The right and left infracolic spaces communicate behind the greater omentum, in front of the mesentery, and through the labyrinth formed by the intestinal coils, but the right space does not communicate so freely with the pelvis as does the corresponding left space. The right infracolic and pelvic spaces do communicate around the inferolateral margin of the cæcum, and in front of the ileocæcal junction, but the lower end of the mesentery, and the cæcal fold (when present), seem to act to a certain extent as barriers between the two spaces. As a consequence, effusions arising in the pelvis ascend more readily into the left than into the right infracolic space. If a Lane's ileal membrane is present, binding down the lower end of the small gut to the posterior abdominal parietes, fluids may spill over more readily from the right into the left infracolic space, and so into the pelvis.

3. The Left Infracolic Space.—This space lies below the distal half of the transverse colon and mesocolon, and on the left side of the mesentery (*Fig.* 185). Anteriorly, laterally, and posteriorly it is bounded by the corresponding portions of the abdominal parietes, and in front it is covered to a variable extent by the greater omentum; the transverse colon and mesocolon overlie the upper part of the space. Its posterior boundary is formed by the left lumbar paravertebral groove below the level of the root of the transverse mesocolon, and, as on the right side, the hollow is partly filled up by such structures as the lower pole of the left kidney, the perinephric and paranephric fat, the psoas and quadratus lumborum muscles, the descending colon, and part of the duodenum. It is wider below than above, due to the obliquity of the mesenteric root, and it contains the greater part of the small intestines. The descending colon lies in the space if it has a mesocolon, but behind it when the latter structure is absent.

There are two grooves alongside the descending colon, the left external and internal paracolic grooves (*Fig.* 185). They are smaller than the corresponding channels on the right side, as the descending is usually narrower than the ascending colon, and they are less often interrupted by small peritoneal folds passing between the colon and the adjacent parietes. The external groove, however, is more or less cut off from the left subphrenic space by the phrenicocolic ligament, extending between the left colic flexure and the diaphragm opposite the tenth and eleventh ribs, but the groove opens into the pelvic cavity below, and effusions may spread upwards or downwards by this pathway.

The left internal paracolic groove may also conduct effusions upwards or downwards between the pelvis and left infracolic spaces. It opens towards the pelvis below, while above it widens out into a fossa bounded superiorly by the left colic flexure, medially by the duodenojejunal junction and upper end of the mesentery, and laterally by the descending colon. The left paramesenteric gutter, the groove on the left side of the mesenteric root, also ends in this space above, and effusions occasionally spread along this pathway—for example, from perforation of an appendix lying in the splenic, promontoric, or pelvic positions.

The left infracolic space communicates freely with the pelvis, and it communicates with the right infracolic space behind the greater omentum, in front of the mesentery, and between the coils of small intestine. Above, it communicates with the left subphrenic space around the edge of the greater omentum and in front of the transverse colon.

4. The Pelvic Space.—The pelvic intraperitoneal space is potential like all the others, and differs in the two sexes. In both male and female the upper portion is partly subdivided by the pelvic mesocolon, and the most dependent part is below the level of the sacral promontory in the supine position, though the depth varies according to the state of distension of the rectum. The postero-inferior boundary is the reflexion of the peritoneum from the anterior rectal wall, but in the male the peritoneum passes to the upper parts of the seminal vesicles and the bladder, while in the female the peritoneum invests the posterior vaginal fornix and most of the uterus before reaching the bladder (*Fig.* 188). The recto-uterine pouch of the female is deeper than the rectovesical pouch in the male, as the peritoneum extends farther down on the rectal wall in the former, but in both sexes the sacrogenital folds form the common upper boundaries. By the sides of the rectum and bladder there are shallow depressions known as the pararectal and paravesical fossæ respectively.

In the female the uterus, broad ligament, and upper part of the vagina divide the rectovesical space into deeper recto-uterine and shallower uterovesical fossæ (*Fig.* 188); the uterine tubes open directly into the former, thus providing an additional route for the spread of infection in the female.

The communications of the pelvis with the main infracolic spaces and with the paracolic grooves have already been described in connexion with the other spaces, and need not be repeated.

EXPERIMENTAL INVESTIGATIONS

A knowledge of the intraperitoneal spaces and communications is essential to an understanding of the spread and localization of intraperitoneal effusions, and as the majority of acute perforations involve the duodenum, stomach, and appendix, the spread of effusions from the areas in which these viscera lie is of most importance. The problem has been studied by various investigators, and it appears that general agreement has been reached, but for reasons already stated it was decided to re-investigate the matter to test the accuracy of the commonly accepted descriptions.

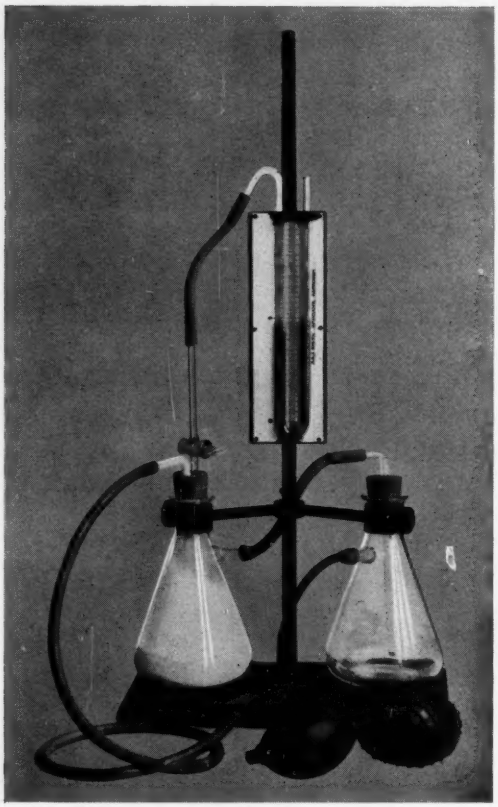


FIG. 189.—A photograph of the simple injection apparatus used in the experiments.

Various dyes were used in early experiments, but they proved to be too diffusible, a defect which was not remedied by the addition of gelatin and agar, and a further attempt, employing a coloured starch solution, was also a failure. It was then decided to use a fine barium emulsion, and to study the spread by means of radiography, and although the first five and several subsequent experiments were failures, it was evident that the method was capable of giving good results.

The final method adopted was simple. The experiments were performed on stillborn infants, *preferably fresh*, or preserved with a watery solution containing 5 per cent carbolic, 5 per cent glycerin, and 5 per cent alcohol, which prevented decomposition for the short period during which the specimens were kept; fœtuses hardened with formalin were found to be useless. Perforations were made in various hollow viscera through which a cannula or needle was passed. A very fine barium emulsion was then injected slowly, under low pressure (10 to 15 mm. Hg), one to three hours being necessary for each experiment. A photograph of the injection apparatus used is shown in *Fig.* 189. The course taken by the injection was observed on the radiographic screen, and in many cases radiographs were taken every 10 to

15 minutes, or oftener if any interesting appearance was detected on the screen. Naturally it is impossible to imitate experimentally the effects produced by intestinal or diaphragmatic movements in life, but apart from hastening the diffusion of fluids, it is unlikely that they have any influence on the actual direction of spread, and so they have no direct bearing on the matter under investigation. At the end of the experiment the pressure was raised to see if the vaginal processes were patent, and afterwards each specimen was dissected in order to check the X-ray appearances, and the femora, tibiæ, and tarsal bones were examined to establish the state of maturity.

In all 48 experiments were performed, but 8 cases were rejected as failures, usually because of leakage of barium, or because it was considered that there had been too much visceral disturbance. To ensure accuracy it was decided that certain conditions were essential, viz.: (1) A small parietal incision; (2) A minimal amount of visceral disturbance and displacement; and (3) Low injection pressures. During the experiment the body was kept supine, as this is the position normally adopted by patients suffering from acute spreading peritonitis.

The second condition was regarded as most important, and it largely decided the form which the experiments ultimately assumed. To begin with, artificial perforations were made in various hollow viscera, but it soon became evident that it was impossible to do so in many cases without creating considerable visceral disturbance, and it might have been objected that the results were therefore valueless. It was found, however, that perforations of the posterior gastric wall could be produced through a small parietal incision with the absolute minimum of visceral disturbance. A small transverse incision was made on the left side, extending $\frac{1}{2}$–$\frac{3}{4}$ in. inwards from the eighth or ninth costal cartilage, and through this opening the antero-superior surface of the stomach was identified, raised up gently with small toothed forceps, transfixed with two linen sutures, and opened. The postero-inferior stomach wall was then picked up, and a needle or small cannula was pushed obliquely through it into the left subhepatic space (lesser sac). Lastly the two sutures were tied around the needle (or cannula), which was further fixed by parietal sutures, but great care had to be taken not to displace the point.

Actually 31 of the 40 successful experiments were performed in this way, and though perforations of the posterior gastric wall are not common, they have the following experimental advantages: (1) There are no serious technical difficulties to overcome in producing them; (2) There is the absolute minimum of visceral displacement and disturbance—in particular there is no interference with the right subphrenic, infracolic, and pelvic spaces; (3) The fluid first fills up the lesser sac, and thus information is obtained about the extent of this compartment; (4) The fluid always escapes (at least in infants where cribriform lesser omenta are rare) through the epiploic foramen into the right subhepatic pouch, and thus simulates exactly an effusion arising in this space. The method therefore kills several birds with one stone, for it gives information about the spread of an effusion from a perforated duodenal ulcer, a perforated posterior gastric ulcer, a perforation of an anterior gastric ulcer near the pylorus, and a perforated gall-bladder, while it also reveals the extent of the left subhepatic space (the lesser sac). The appendix apart, these lesions are the commonest causes of spreading peritonitis.

The remaining 9 experiments were of the following nature. In 5 cases a cannula was introduced into the appendicular lumen through a cæcal incision, and an

artificial perforation was produced by removing the tip of the appendix. In 2 cases the anterior rectal wall was pierced by a needle inserted above the line of peritoneal reflection, and in 2 cases a needle was passed through the posterior vaginal fornix into the recto-uterine pouch.

The appendicular operation, while technically not difficult, did not give results on which great stress could be laid, as in every case the position of the organ and of its surroundings was disturbed. Thus the most important experimental condition was violated, and that is why only 5 experiments of this type were performed.

In 2 cases the perforated appendix was placed artificially in the paracolic position, and the effused barium spread up the right external paracolic groove to invade the right subphrenic space and the posterior part of the right subhepatic space. It also invaded the right infracolic space very early, and later spread from

A B

FIG. 190.—The spread of a peritoneal effusion from the subhepatic spaces. A, A cannula has been passed into the left subhepatic space (lesser sac) and the injection of barium has just commenced. B The left subhepatic space is almost full.

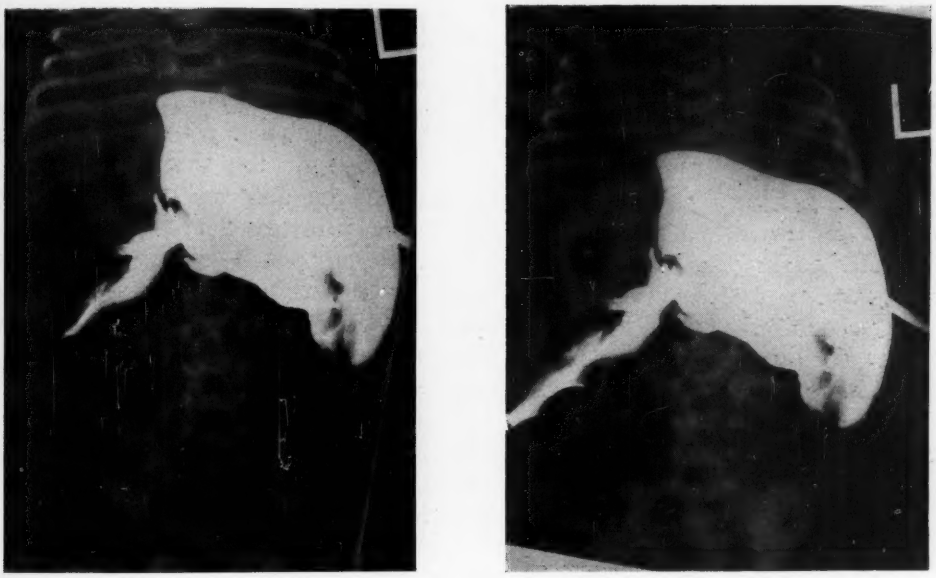
there into the left infracolic space, and so to the left subphrenic space. The pelvis was invaded at a fairly late stage, and the spread occurred in two ways. Some barium escaped around the lower cæcal margin and overflowed into the pelvis, but a larger amount reached it from the left infracolic space. Throughout the experiment the body was kept in the recumbent position, and doubtless the pelvis would have been invaded much earlier had the semi-recumbent position been employed.

In 3 cases the perforated appendix was artificially placed so that its tip was directed downwards and inwards towards the sacral promontory or into the pelvis. The barium diffused rapidly both into the pelvis and into the left infracolic space, and spread from the latter into the left subphrenic and right infracolic spaces, and thence to the right subphrenic and right subhepatic spaces. There was no evidence that the barium selected the paracolic grooves as convenient channels of spread—

it was more a matter of gradual diffusion throughout the peritoneal cavity. The left subhepatic space (lesser sac) remained clear throughout in all 5 experiments.

The manner of spread of the barium in the 4 cases where the rectum or posterior vaginal fornix was perforated was very similar to the above, except that in one case of rectal perforation a streamlet of barium passed upwards along the left external paracolic groove until it was arrested by the phrenicocolic ligament.

In one case a cannula was inserted into the uterus and barium was forced in. On raising the pressure to about 100 mm. Hg, the barium began escaping from both uterine tubes into the peritoneal cavity, and it was interesting to note that on the right side a stream of barium entered the right external paracolic groove, apparently before the pelvis was completely filled, and spread upwards towards the right subphrenic space. The upward spread was delayed, but not completely arrested, near



A B

FIG. 191.—The spread of a peritoneal effusion from the subhepatic spaces. A, The barium has escaped through the epiploic foramen into the right subhepatic space (Morison's pouch). B, The right subhepatic space is filling up and barium has just begun to escape from the space by passing in front of the transverse colon.

the right inferior margin of the liver by the presence of a crescentic peritoneal fold stretching from the flank to the outer side of the ascending colon ; this was established at the subsequent dissection. This experiment confirmed the well-known clinical finding that women suffering from a ruptured tubal pregnancy sometimes complain of shoulder pains at a relatively early stage, and doubtless the proximity of the tube to the pelvic brim explains the ready escape of blood, etc., into the paracolic groove. The experiment was regarded as a failure (and is not included in the series) because the pressure used was very high, and the barium persisted in leaking backwards through the vagina.

Taken as a whole, this group of experiments confirmed the statements made by most writers about the spread of an effusion arising from the appendix or from a pelvic viscus.

The experiments, however, designed to study the spread of an effusion from the right subhepatic pouch, gave results entirely at variance with those generally accepted. In this group of 31 experiments the posterior gastric wall was perforated in the manner described, and barium was then injected slowly (*Fig.* 190, A). It first filled up the left subhepatic space (the lesser sac) (*Fig.* 190, B), and then escaped, through the epiploic foramen, into the right subhepatic space (Morison's pouch) (*Fig.* 191, A). Filling up this space, it overflowed in every case in a characteristic manner in front of the transverse colon, usually through the interval bounded by the quadrate lobe of the liver above, the fundus of the gall-bladder on the right side, the falciform ligament on the left side, and the transverse colon below (*Fig.* 191, B). This quadrangular interval is an important site of communication between the right subhepatic space and the antero-internal angle of the right subphrenic space, but it is interesting to note that this was the only part of the latter space which was invaded directly. The remainder of the right subphrenic space was apparently sealed off by some type of valvular action produced by the apposition of the sharp inferior liver margin and the abdominal wall—probably as a result of increased pressure within the right subhepatic pouch—and the radiographs show that the main part of the right subphrenic space was not involved until the barium spread upwards along the right external paracolic groove.

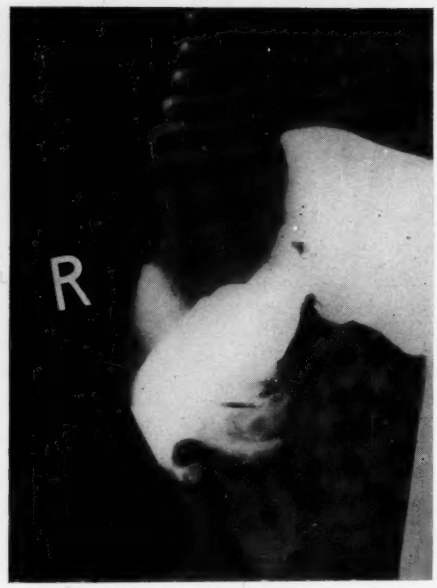
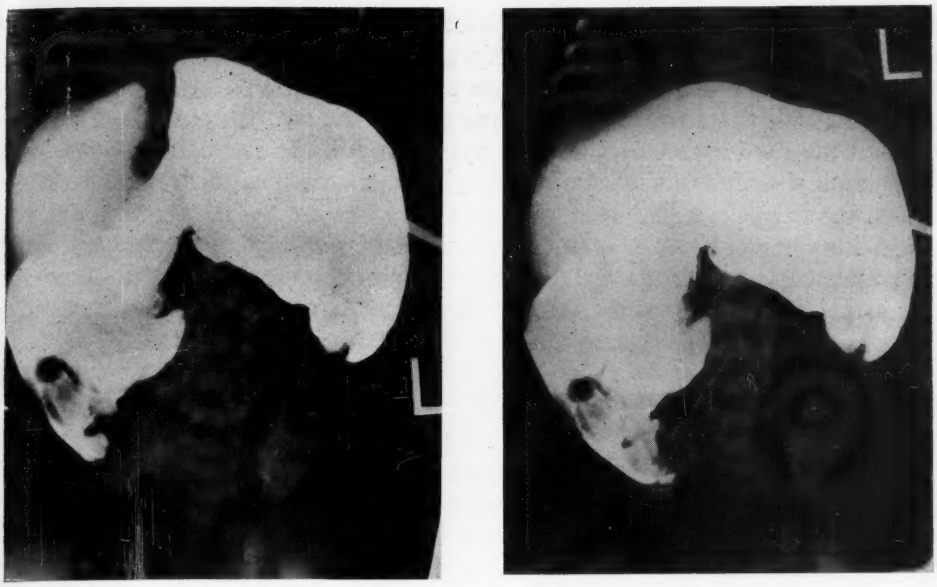


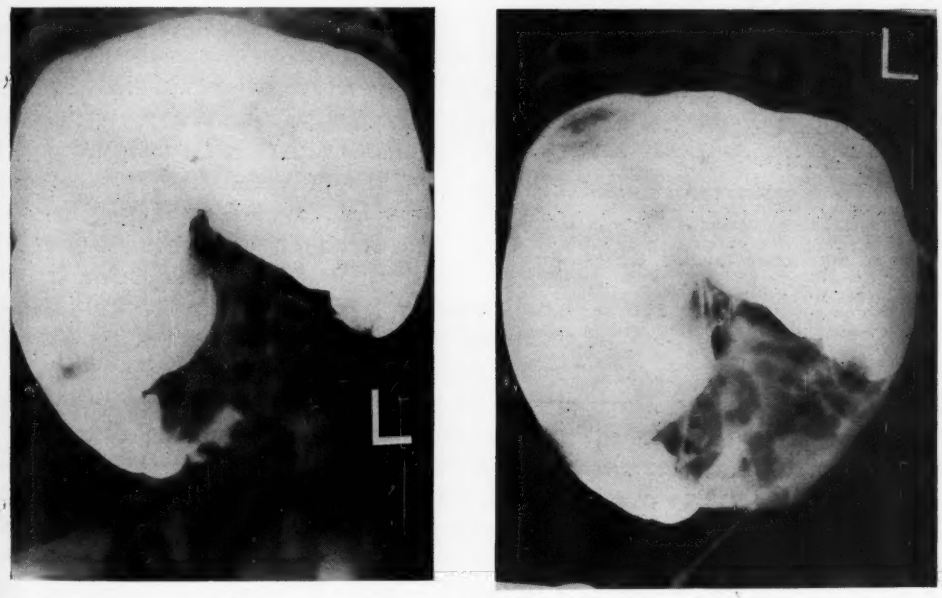
FIG. 192.—The spread of a peritoneal effusion from the subhepatic spaces. The barium has escaped from the right subhepatic space into the right infracolic space in the manner described in the text, and has spread downwards as far as the ileocæcal region. Some of the barium has passed in front of the lower part of the ascending colon, and is beginning to spread *upwards* along the right external paracolic groove.

The barium, having escaped from Morison's pouch in the manner described, spread downwards and outwards in front of, or between, the coils of small intestines in the right infracolic space, and so reached the ileocæcal region. It also flowed over the front of the lower part of the ascending colon and filled up the lower part of the right external paracolic groove (*Fig.* 192). Thereafter it spread *upwards* along this groove, and invaded the right subphrenic space (*Fig.* 193, A, B). In 6 cases the upward flow was arrested about the level of the right inferior liver margin by a fold of peritoneum passing between the flank and the ascending colon, forming a barrier across the groove. Sometimes the barium as it accumulated overtopped this barrier, just as a flooded dam overflows its sluice, and entered the groove beyond ; in other cases the upper part of the groove was invaded by barium passing across the front of the ascending colon above the level of this peritoneal fold. The fact that a peritoneal fold was the cause of the obstruction was verified in each case by subsequent dissection. Occasionally there were minor hold-ups due to the presence of smaller peritoneal folds. Some of the barium spilled over the ileocæcal junction and around the lower margin of the cæcum and so involved the left infracolic space (*Fig.* 194, A). From there the barium passed



A B

FIG. 193.—The spread of a peritoneal effusion from the subhepatic spaces. A, The barium in the right external paracolic groove has now invaded the right subphrenic space. The right infracolic space is becoming extensively involved, and some of the barium is permeating the labyrinth of intestinal coils to enter the left infracolic space. A small stream of barium is seen passing downwards around the inferolateral cæcal margin. B, The right subphrenic space is almost completely filled, and the small streamlet of barium passing downwards around the inferolateral cæcal margin has been arrested temporarily at the pelvic brim; at this point it is below the lower end of the mesentery and so is actually in the left infracolic space. The spread from the right to the left infracolic space continues.



A B

FIG. 194.—The spread of a peritoneal effusion from the subhepatic spaces. A, The spread from the right to the left infracolic space is a little further advanced. It is interesting to note that only a part of the streamlet of barium which trickled round the cæcum into the right lower angle of the left infracolic space actually enters the pelvis; the remainder spreads upwards amongst the coils of intestine in the left infracolic space. B, All the intraperitoneal spaces are now extensively involved, but it is noteworthy that even at this late stage the pelvis remains relatively clear.

both upwards to involve the left subphrenic space and downwards to invade the pelvis, which was involved last of all (*Fig.* 194, B).

Sometimes the left subphrenic space was invaded at an early stage by barium passing through the communication (bounded by the free margin of the falciform ligament, the transverse colon, and the pyloric end of the stomach) between it and the right subphrenic and subhepatic spaces, but in no case was there massive involvement of the left subphrenic space through this channel.

In interpreting films of stillborn infants it is necessary to remember that the small intestines are often filled largely with air and the colon with meconium. Unless this fact is appreciated, the air-containing interval seen above the barium stream in *Fig.* 191, B, for example, may readily be regarded as the right colic flexure and proximal part of the transverse colon, whereas it is really produced by coils of small intestines. The barium did *not* reach the right external paracolic groove by passing above the hepatic flexure in a single case in this series of 31 experiments, and it was noted that a hepatorenal ligament was not invariably present. This finding proves that there must be other factors, in addition to the hepatorenal ligament, tending to prevent the passage of fluids from the right subhepatic space into the right external paracolic groove, and the possible factors are discussed later.

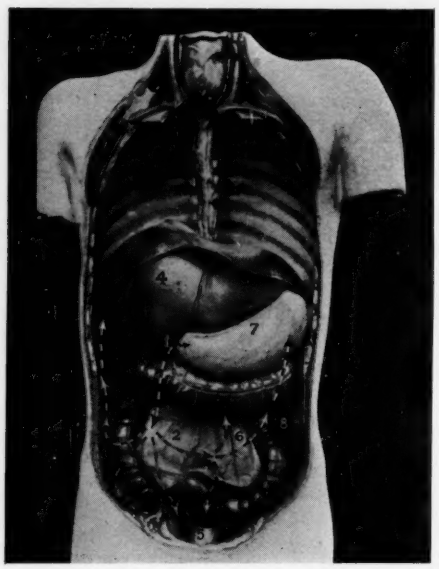


FIG. 195.—Showing the lines of spread of an effusion arising in the right subhepatic space. 1, Exit from right subhepatic space, bounded by the gall-bladder, the quadrate lobe of liver, the free edge of the falciform ligament, and the transverse colon; 2, Right infracolic space; 3, Right external paracolic groove; 4, Right subphrenic space; 5, Pelvic space; 6, Left infracolic space; 7, Left subphrenic space; 8, Left external paracolic groove.

To summarize: An effusion escaping from a perforated duodenal ulcer, gall-bladder, or posterior gastric ulcer, or from an anterior gastric ulcer near the pyloric end of the stomach, invades the right subhepatic space. The fluid collecting in the space overflows into the antero-internal angle of the right subphrenic space, usually through a quadrangular interval bounded by the quadrate lobe of the liver, the gall-bladder, the falciform ligament, and the transverse colon. It does *not* invade the main part of the right subphrenic space at this stage, however, but spreads downwards and to the right in front of the proximal part of the transverse colon, and around the margin of the greater omentum, and so invades the right infracolic space. Some fluid flows over the ascending colon into the lower part of the right external paracolic groove, and then gravitates *upwards* along this groove to invade the right subphrenic space, while the rest slowly permeates the interspaces between the coils of intestine in the right infracolic space. It then escapes into the left infracolic space by passing in front of the ileocæcal junction, and, spreading gradually throughout the space, it extends upwards to involve the left subphrenic space and downwards to invade the pelvis. Sometimes the pelvis is also invaded by a small streamlet which trickles around the lower cæcal margin and runs over the pelvic

brim, but *it is noteworthy that, in the recumbent position, the pelvis remains free at a relatively late period.* The left subphrenic space is often slightly contaminated at an early stage by barium passing into it beneath the free margin of the falciform ligament and above the transverse colon and mesocolon, but no massive invasion occurs by this channel. The manner of spread is shown in *Fig.* 195.

DISCUSSION

As the results of the experiments on the spread of an effusion from a perforated appendix or pelvic viscus confirm generally accepted views on the subject, no time need be wasted in discussing them.

The results of the other experiments, however, are diametrically opposed to generally accepted views, and so they merit discussion. It is probably true to say that most surgeons believe that an effusion arising in or near the right subhepatic pouch spreads in the manner indicated by Livingston (1932) in his well-known diagram, showing the lines of communication in spreading peritonitis (*Fig.* 196). In the text Livingston states: "The several abdominal drainage basins communicate more or less freely with one another. For example, free fluid from a perforated gastric ulcer may first invade the subhepatic space or the omental bursa,* then spread upward to the right subphrenic area, or again extend downward along the right paracolic gutter (there to simulate the peritonitis associated with a perforation of the appendix); it may even pass onwards into the pelvis, and when sufficient accumulation has occurred, the fluid may well upward out of the pelvis to involve either subcolic fossa or to pass into the left paracolic groove, to extend farther upward into the perisplenic space. A spreading peritonitis, as a spreading infection of the hand, travels onward by way of fairly fixed channels."

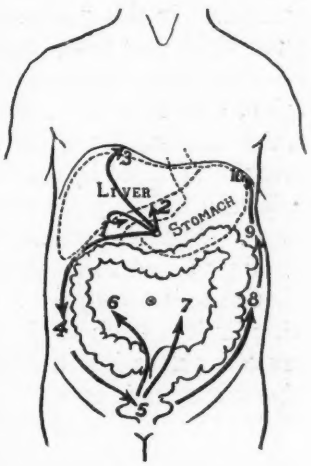


FIG. 196.—"Spreading peritonitis. Communication of the 'drainage basins' of the peritoneal cavity" (*Livingston*). The lines of spread indicated here are those hitherto generally accepted and described. 1, Right subhepatic space; 2, Left subhepatic space (lesser sac); 3, Right subphrenic space; 4, Right external paracolic gutter; 5, Pelvic space; 6, Right infracolic space; 7, Left infracolic space; 8, Left external paracolic groove; 9, Perisplenic space (a subdivision of the left subphrenic space); 10, Left subphrenic space. Compare wtth *Fig.* 197.

Other writers express somewhat similar views.

Box (1910): "The right kidney well† may be capable of holding a pint of fluid before it overflows into other parts of the peritoneal cavity. When it does overflow it overflows in certain definite directions. The easiest channel when the pouch has been filled from above is along the right external paracolic groove towards the pelvis."

Moynihan (1926): "A duodenal ulcer may perforate at once and acutely into the peritoneal cavity, or may slowly destroy all the coats of the bowel and lead to the formation of a localized encysted abscess. If the former, the fluid escaping

* Left subhepatic space or lesser sac.
† Morison's pouch, or right subhepatic space.

from the viscus is free to run at large in the peritoneal cavity. In many of the cases, as is seen from a study of the records, a well-defined path is taken. The fluid (generally mucus, more or less tinged with bile) escapes on to the upper surface of the transverse mesocolon to the right of the hillock which is formed by the fitting in of the transverse colon to the greater curvature of the stomach. It therefore tends to run to the right of the hepatic flexure, and then to descend along the outer side of the ascending colon to the iliac fossa. There collecting, it may cause symptoms strongly suggestive of appendicitis. From the iliac fossa the fluid drains to the pelvis, and, filling that, overflows into the left iliac fossa."

Bailey (1931): "When a duodenal ulcer perforates, the ascending colon acts as a watershed and directs the escaping fluid to the right iliac fossa." (The diagram given to illustrate the manner of spread shows the fluid passing above the right colic flexure, and then down the right external paracolic groove to the pelvis.)

Blake (in *Nelson Loose-Leaf Surgery*): "Beneath the liver, between it and the kidney, is a pocket in close relation with the pyloric portion of the stomach, the duodenum, the gall-bladder, and the right kidney. Extravasations in this region accumulate in this pocket and then flow to the right down the right lumbar gutter to the right iliac fossa, and finally to the true pelvis."

Lake and Marshall (1934): "Fluid effused in the anterior gastroduodenal region, e.g., from a perforated ulcer or gall-bladder, is prevented from descending on the deep surface of the abdominal parietes by the apposition to this of the transverse shelf formed by the transverse colon and great omentum: it collects in quantity under the diaphragm, under and over the liver, and overflows into the right kidney pouch. Thence, particularly if still aided by gravity, it passes down the loin to the iliac fossa; and so to the pelvis."

Cokkinis (1936): ". . . duodenal perforations are frequently subacute, leakage being limited to the right renal pouch, and spreading from this along the external paracolic sulcus into the right iliac fossa and pelvis, or round the liver edge to the subdiaphragmatic space."

The above may be considered as representative opinions, and the ideas expressed may be summarized as follows:—

1. Fluid effused from the perforated duodenal ulcer, gall-bladder, etc., first fills up the right subhepatic pouch, which may hold about a pint before overflowing.
2. From there it spreads upwards to involve the space lying between the right lobe of the liver and the diaphragm (right subphrenic space).
3. It also spreads outwards to the right external paracolic groove, and then downwards along it to the ileocæcal region.
4. From the right external paracolic sulcus it invades the pelvis.
5. Filling the pelvis, it wells upwards to involve either infracolic space.
6. It may also pass from the pelvis into the left external paracolic groove and spread upwards along this groove to the left subphrenic space (which includes the perisplenic space).

The experimental findings, and observations made in the operation theatre and dissecting room, negative a number of these suggestions, and they may be considered *seriatim.*

1. *Fluid effused from a perforated duodenal ulcer, gall-bladder, etc., first fills up the right subhepatic pouch, which may hold about a pint before overflowing.*—Fluid effused from the common type of acute duodenal perforation, from perforation of

the gall-bladder, and from perforation of an anterior gastric ulcer near the pyloric end, invades the right subhepatic space directly, while fluid escaping from an acute perforation of a posterior gastric ulcer first invades the left subhepatic space or lesser sac, and then spreads into the right subhepatic space through the epiploic foramen. This space is filled up more or less rapidly, depending on the rate of extravasation. On these points there is no disagreement, but it is doubtful if the pouch will hold one pint without overflowing unless some of the communications with other spaces are obliterated by adhesions, and in most cases of acute perforation such adhesions are absent. Many of the structures helping to bound the space are relatively solid or immobile (liver, kidney, suprarenal, pancreas, right colic flexure, and diaphragm) and are not readily displaced. Thus fluid accumulating in the pouch tends to displace the other boundaries—particularly the transverse colon and pyloroduodenal junction, which help to form the postero-inferior boundary. They are pressed downwards and backwards, and, as a result, fluid spills over the front of the transverse colon into the right infracolic space long before a pint of fluid is present in the space. This can readily be tested during laparotomy. If a tube is placed in Morison's pouch and sterile saline injected through it, the fluid will well upwards from the pouch long before a pint has been injected.

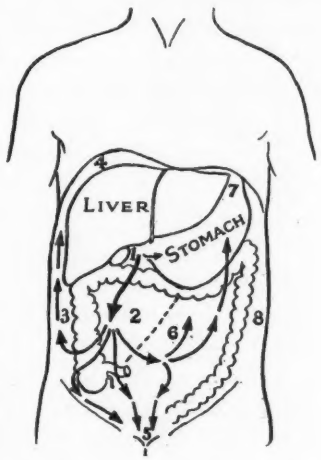


FIG. 197.—Diagram of spreading peritonitis. The lines of spread suggested by the writer. The dotted line represents the attachment of the mesentery. 1, Exit from right subhepatic space, bounded by the inferior border of the quadrate lobe of the liver, the free edge of the falciform ligament, the fundus of the gall-bladder, and the transverse colon; 2, Right infracolic space; 3, Right external paracolic groove; 4, Right subphrenic space; 5, Pelvic space; 6, Left infracolic space; 7, Left subphrenic space; 8, Left external paracolic groove. Compare with *Fig.* 196.

2. *The fluid, having escaped from Morison's pouch, spreads upwards to involve the right subphrenic space.*—This is doubtful on clinical and experimental grounds. The presence of an irritating fluid in contact with the diaphragm gives rise to referred pain in the shoulder region, yet pain of this type is an uncommon symptom, at least in the earlier stages of a perforated duodenal ulcer. One has seen a number of patients with pain and tenderness in the right iliac fossa due to a spreading peritonitis from a perforated duodenal ulcer who made no complaint of right shoulder pain, and denied they had any when a leading question was put to them.

Further, in this series of 31 experiments in which the spread from the right subhepatic space was studied, it was found that the right subphrenic space, with the exception of a small area at the antero-internal angle, was *not* invaded in the early stages. It was discovered that fluid in the right subhepatic pouch soon overflowed into the right infracolic space and spread downwards and outwards to the ileocæcal region. Some passed over the ascending colon into the right external paracolic groove, and then spread *upwards* along the groove and entered the right subphrenic space (*Fig.* 197). The involvement of the right subphrenic space is therefore indirect and not direct.

It is interesting to speculate why the right subphrenic space escapes direct invasion, since it apparently communicates so freely with the right subhepatic pouch around the inferior margin of the liver. There may be two main factors at

work: (1) The pressure produced by fluid accumulating in the right subhepatic space forces the boundaries apart. The liver is pushed upwards and forwards, and its thinnish inferior margin may have a valvular action when pressed against the abdominal parietes, preventing the influx of foreign materials into the subphrenic space (*Fig.* 198). (2) This same pressure displaces the transverse colon downwards, and allows the escape of fluid from the pouch anteriorly. With the body in the recumbent or semi-recumbent position, the fluid as it flows out in this situation lies below the level of the interval between the liver and diaphragm in front, and the action of gravity directs the fluid away from the subphrenic space.

3. *Fluid spreads outwards to the right external paracolic groove, and then downwards along it to the ileocæcal region.*—The writers who discuss this point, or who show the lines of spread diagrammatically, all agree that the fluid escapes into the external paracolic groove through the interval between the liver, right kidney, and right colic flexure. It is often stated that the right subhepatic space communicates directly with the right external paracolic groove, and some even describe the groove as being a direct extension downwards of the space. This conception is inaccurate, as the space and groove are often almost completely separated by the hepatorenal ligament, and even when this peritoneal fold is absent, the space between liver, kidney, and colic flexure provides no easy pathway for the escape of fluid. The matter is easily tested in the operating theatre or dissecting room. It is very easy to enter the right subhepatic space from the front, between the liver and transverse colon, but not so easy to pass the fingers through the interval between the liver, right kidney, and right colic flexure, because two of the boundaries are relatively solid and immobile, and the hepatorenal ligament bridges the opening more or less completely.

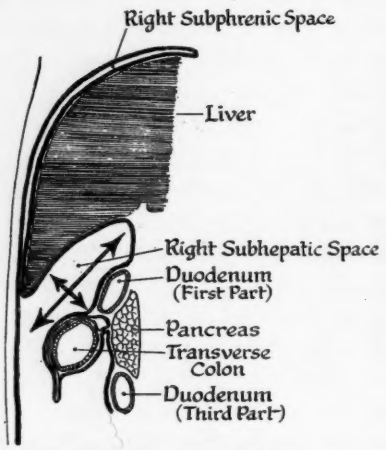


FIG. 198.—Diagram showing the suggested mechanism which may prevent the spread of an effusion from the right subhepatic to the right subphrenic space. The fluid collecting in the former space tends to push the boundaries apart, and a valvular action may be produced by the apposition of the thin inferior liver margin and the abdominal parietes.

The position therefore arises that fluid in the right subhepatic space cannot readily escape into the right external paracolic groove because of the hepatorenal ligament and the close apposition of relatively solid or immobile structures such as the liver, kidney, and right colic flexure; it cannot escape into the left subhepatic space (lesser sac) because the epiploic foramen is a mere slit offering considerable resistance to the passage of fluid; it cannot escape upwards or downwards because of the liver above and the transverse colon and mesocolon below; and so it finds its easiest mode of egress antero-inferiorly between the liver and transverse colon, a mode of exit which is favoured by the relative shortness or absence of the transverse mesocolon at this point. At first sight it may appear that this manner of exit means that the fluid is flowing directly against the force of gravity, but further consideration will show that this effect is more apparent than real. The pressure within the space pushes the colon downwards and backwards, and also tends to flatten it, so that it may actually lie about the same level (with the body supine) as the pyloroduodenal junction or the neck of the gall-bladder. The fluid therefore escapes over a gently

sloping platform, and even if it has to flow slightly uphill, the force required to accomplish this may be considerably less than that which would be necessary to force a passage through the narrow straits between the liver and right kidney. The experimental findings support these theoretical considerations, for it has been shown that fluid accumulating in Morison's pouch escapes into the right infracolic space through the interval between the liver, gall-bladder, falciform ligament, and transverse colon, and not into the right external paracolic groove through the potential channel between the liver, right kidney, and right colic flexure.

Even if the fluid did escape into the paracolic groove in the way suggested it would not run downwards to the ileocæcal region, because the groove slopes upwards, the lower transverse barrier formed by the pelvic brim being distinctly higher than the upper half of the right paravertebral fossa, though of course the slope is diminished by the presence of the right kidney, right suprarenal gland, etc., which partly fill up the deepest part of the fossa. Thus if the fluid extended downwards along the paracolic groove it could do so only by a process of accumulation, just as a loch, fed by flood waters, gradually encroaches on the surrounding land. Besides, the groove is often interrupted by peritoneal folds which interfere with the free flow of fluid.

4. *From the right external paracolic sulcus it invades the pelvis.*—Undoubtedly this may happen, but it is wrong to suggest that it is the usual method of pelvic invasion. As a rule the fluid escapes from the right into the left infracolic space by passing in front of the ileocæcal junction, and the pelvis is invaded from the latter space. With the body in the recumbent position the pelvis is usually involved fairly late, and often the left infracolic space is extensively permeated before the fluid escapes into the pelvis. Experimentally the streamlet of barium which occasionally escaped around the infero-lateral cæcal margin into the pelvis was generally insignificant in volume compared with the relatively large inflow from the left infracolic space.

5. *Filling the pelvis, it wells upwards to involve either infracolic space.*—From what has been said already it is clear that this is inaccurate, and would not be correct even if the patient could be placed in the Fowler position at the very onset of a perforation—an eventuality which must be so rare as not to merit consideration. Therefore if the pelvis is involved in an acute spreading peritonitis due to a perforated viscus in the supracolic region of the abdomen, it can be taken as certain that the infracolic spaces will also be involved to a greater or lesser degree. Theoretically some may argue that direct pelvic involvement from an effusion in the right subhepatic space might occur if the greater omentum was so voluminous that it covered the small intestines and ascending and descending colons completely, and so directed fluids straight downwards from the supracolic region to the pelvis. There are two obvious objections to this suggestion: firstly, such an omentum must be a great rarity; and, secondly, even if it did exist in a person who had developed an acute perforation, this type of catastrophe forces the patient to adopt the supine position, and the fluid would then be directed over the omentum towards the flanks and not the pelvis. In other words, the external paracolic grooves would be the sites of collection of the effusion, and they are subdivisions of the infracolic spaces.

6. *It may also pass from the pelvis into the left external paracolic groove, and spread upwards along this groove to the left subphrenic space.*—This contention is also negatived by the experimental findings, which showed that the left subphrenic space

is invaded by fluid spreading upwards, around the left margin of the greater omentum and over the transverse colon, from the left infracolic space. In one case barium was detected running upwards along the left external paracolic groove, but that was in a specimen where the retrovesical pouch had been perforated through the rectum, and where the pelvis filled up with barium before any other space was invaded; and even then, the way to the left subphrenic space along this channel was blocked by the phrenicocolic ligament. It was also shown that the left subphrenic space may be invaded in the early stages by fluid spreading from Morison's pouch through the interval between the free edge of the falciform ligament, the pyloric end of the stomach, and the transverse colon, but this cannot be a free pathway of communication, for in no case was gross contamination of the left side produced in this way.

SUMMARY

1. A description is given of the intraperitoneal barriers and drainage basins.

2. The importance of secondary barriers in the form of peritoneal folds and viscera is emphasized.

3. It is suggested that an understanding of the divisions of the supracolic space would be simplified if a uniform terminology were adopted. The terms 'right and left subphrenic' and 'right and left subhepatic' have much to commend them.

4. The extent of the lesser sac in 31 cases is described.

5. The communications by which effusions may spread from one space to another are considered.

6. Experiments are described which had for their object the tracing of the lines of spread of acute intraperitoneal effusions, particularly those arising in the right subhepatic space (Morison's pouch), and it is shown that the generally accepted teaching is inaccurate.

7. An effusion accumulating in the right subhepatic space does not normally escape between the liver, right kidney, and right colic flexure into the right external paracolic groove, but overflows into the right infracolic space through the interval between the liver, gall-bladder, transverse colon, and falciform ligament.

8. The right external paracolic groove, which is really a subdivision of the right infracolic space, is involved later by a spread across the front of the ascending colon, and the fluid then runs *upwards* along this groove and so invades the right subphrenic space; this space, except for a small area at the antero-internal angle, is *not* invaded by direct spread from the right subhepatic space, and the possible reasons for this are discussed.

9. The pelvis is mainly invaded by a spread of fluid from the infracolic spaces, and there is no experimental evidence that the main channel of invasion is along the right external paracolic sulcus.

10. Similarly it is shown that the common belief that fluids spread from the pelvis to the left subphrenic space along the left external paracolic groove cannot be verified experimentally, as the fluid is arrested by the phrenicocolic ligament. The left subphrenic space is involved by direct spread from the main left infracolic space (the left external paracolic groove is really a part of the left subphrenic space), and to a lesser extent through the communication which exists between the right and left subhepatic spaces in the interval between the pyloric end of the stomach, the transverse colon, and the free edge of the falciform ligament.

I am deeply indebted to Professor A. Low, Emeritus Professor of Anatomy, Aberdeen University, who allowed me, over a period of years, to make free use of the X-ray equipment in the Anatomy Department; and I wish to record my thanks to him, to Professor J. R. Learmonth, Professor of Surgery, Edinburgh University, and to Professor R. D. Lockhart, Professor of Anatomy, Aberdeen University, for much valued criticism and advice. I wish also to place on record my debt of gratitude to the late Dr. A. C. Fowler, Radiologist, without whose tuition and guidance these experiments could never have been begun. The expenses of the investigation have been largely defrayed with the aid of grants from the Medical Research Council.

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SUPERNUMERARY AND ECTOPIC URETERS

CARCINOMA OF A DOUBLE KIDNEY, WITH ECTOPIA OF ONE URETER

By G. H. BAINES

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THE case reported was admitted to Mr. Bernard Ward's Clinic at the Queen Elizabeth Hospital, Birmingham. Tumours occurring in double kidneys are sufficiently uncommon to be of interest, and may present special difficulties in diagnosis. I have been able to find records of only 14 growths of various types occurring in double or supernumerary kidneys.[1, 2, 3, 4, 18]

The case reported was further complicated by ectopia of one ureter and hydronephrosis of the corresponding half of the kidney.

Supernumerary Ureters.—Supernumerary ureters are not uncommon. They occur in 3 to 4 per cent of cases whether investigated by pyelography,[8] or post-mortem examination.[11, 12, 13] They are found two[18] or three[9] times more often in women than in men; 80 per cent are unilateral,[14, 18] and only 30–60 per cent are complete.

The importance of the condition lies in their very great liability to obstruction and infection.[3, 8, 10, 15, 16] These complications may come on relatively early in life, often in the third decade.[8] A high incidence of ureteral stricture has been noticed.[10]

It is probable that in the majority of cases the first complication is obstruction producing some degree of hydronephrosis, and that this may remain symptomless until infection supervenes. It is important to remember the possibility of supernumerary ureters when investigating cases of obscure hæmaturia or pyuria.

Ectopic Ureters.—Ectopic ureters are very much less common. A large proportion of them are associated with supernumerary ureters. In accordance with Weigert's law the ureter draining the upper part of the kidney opens lower in the urinary tract, and it is always this orifice which is ectopic. Malgras,[6] however, asserts that this is not always so. In the case here reported the law holds good.

The developmental abnormalities concerned in the production of ectopic ureters have been well worked out. They may be grouped under three main headings[20]:—

1. Defective absorption of the terminal portion of the Wolffian duct, or failure of the normal migration of the ureteric orifice. The ureter may then terminate in the urethra, ejaculatory ducts, seminal vesicles, vas deferens, vestibule of the vagina, Gartner's duct, or an abnormal portion of the bladder.

2. Abnormal shifting of the ureteric orifice, when it may open into the prostatic utricle, vagina, uterus, or Fallopian tube.

3. Maldevelopment of the urorectal septum, when the ureter may open into the rectum or a cloaca. Other gross developmental abnormalities are usually present in these cases.

Although the ureter may open in any of the above sites, certain of them are by far the commonest. Thom,[18] in a series of 178 cases, gives the following incidence: urethra 70 cases, vestibule of vagina 45, vagina 32, seminal vesicles 17. This agrees with the findings of other writers.

Ectopic ureters occur rather more frequently in females. In males they are usually symptomless and escape detection, but in women a large proportion are diagnosed during life. Sargent[19] found that only 6 out of 63 cases in the male had been discovered clinically, whereas 94 out of 123 cases in the female had been found.

Incontinence associated with normal micturition is the characteristic symptom. This is almost invariably absent in men, as the ectopic opening is above the external sphincter. There are then no symptoms apart from the complications of obstruction or infection. In women the majority of ectopic ureters open below the vesical sphincter and incontinence is present.

Ectopic orifices are almost constantly associated with other abnormalities, congenital or acquired. This is due in the majority of cases to stenosis of the opening. In some their exposed positions must render them peculiarly liable to infection. Some writers[9] go so far as to say that the corresponding kidney "is always hydronephrotic". Thom found a normal upper tract in only 4 of his 178 cases. There seems to be general agreement among surgeons that the kidney concerned is of little functional value.

Diagnosis is often extremely difficult. In the presence of typical symptoms search should be made till the orifice has been found, and a retrograde pyelogram demonstrates the true state of affairs. In males, where symptoms are rare, diagnosis is unlikely, since the kidney corresponding to the ectopic ureter is generally functionless by the time examination is carried out, and it is unusual to find any excretion shadow with an intravenous pyelogram. Sometimes the outline of the lower pelvis may be sufficiently characteristic to raise suspicions of a double kidney. This is especially so if a large renal shadow can be seen above the lower pelvis. In the presence of an unexplained pyuria or hæmaturia posterior urethroscopy should be carried out, as about two-thirds of all ectopic ureters open in this region.

A wide variety of operations have been described for the condition. There is fairly general agreement that heminephrectomy is the best course if possible. Ureterectomy is also advisable, as the dilated stump may act as a reservoir of infection.

CASE REPORT

A man of 51 was admitted with a history of intermittent hæmaturia for eighteen months. The attacks recurred every month or two, and usually lasted about a week. They were associated with penile pain on micturition, and an increase in frequency of micturition (D/N = 8/4). Between the attacks there was at first no frequency, but latterly he had had some increase (D/N = 6/2). The blood was at first intimately mixed with the urine, but for the last four months there had been clots. Since the first attack of hæmaturia there had been pain of an aching character in the right loin, but he had had no colic. For three months he had weakness, dyspnœa on exertion, and a feeling of exhaustion. It was this that sent him to his doctor.

There was nothing of importance in his previous or family history. He had never had any difficulty of micturition, dribbling, or incontinence. He had never noticed any polyuria, or micturition in two parts.

ON EXAMINATION.—There were signs of a gross anæmia. There was marked fullness of the right loin. On palpation a firm smooth mass about the size of a melon was felt. This moved with respiration, and was easily ballotted between the hands. Nothing else of significance was noted. While in hospital he had occasional severe hæmaturia, and at one time passed a tubular blood-cast about a foot long—presumably from his ureter.

The following investigations were carried out :—

Blood-count : R.B.C. 2,840,000 ; Hb 35 per cent.

Blood-urea : 55 mg. per cent.

Urea-concentration Test (MacLean) :—

1st specimen	..	90 ml.	contains	0·9	per cent	urea.
2nd „	..	90 ml.	„	1·5	„	„
3rd „	..	100 ml.	„	1·6	„	„
4th „	..	28 ml.	„	1·8	„	„

Catheter Specimen Urine : Pus ±. R.B.C. ++. Stained films—Gram-positive diplococci ±, Gram-positive bacilli −, Gram-negative bacilli ±. Culture, *Staphylococcus albus* +.

Intravenous Pyelography (Dr. Black).—"Right kidney enlarged. There is poor excretion on the right. Filling defect in the lower calix area, with deformity. Upper and middle calices not filled. Fair excretion on the left side, no definite deformity."

Cystoscopy.—Bladder healthy. Right and left ureteric orifices normal. Catheter passed up the right ureter with ease.

Retrograde Pyelography (*Fig.* 199).—"Confirms the severe deformity of the right pelvis. Ureter normal size."

Radiography of Chest.—"Increased striæ markings in both lungs. No definite secondary deposits."

A diagnosis of carcinoma of the right kidney was made, and operation was decided upon in view of the rapidly increasing anæmia. It was thought that the tumour was removable, and there was no evidence of secondary deposits.

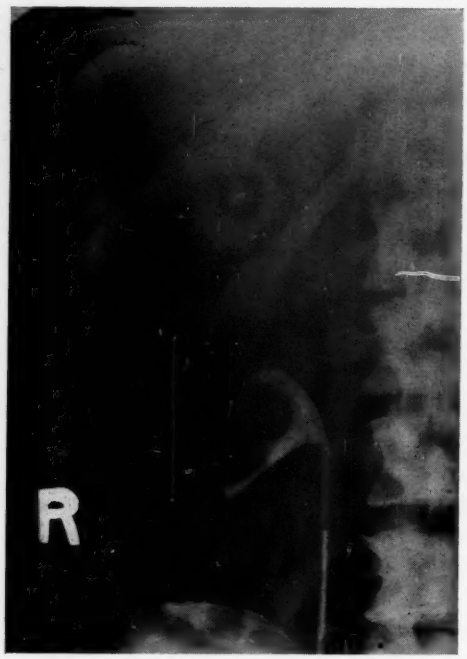

FIG. 199.—Retrograde pyelogram, right kidney.

AT OPERATION.—After careful preparation, including multiple transfusions, nephrectomy was carried out by Mr. Bernard Ward.

There was considerable hæmorrhage from the many large veins in the fat around the kidney. Owing to the great size of the tumour, exposure of the pedicle was impossible and it had to be isolated by touch. It was felt to be invaded by growth. It was impossible to get clear of this, and the pedicle had to be clamped through invaded tissue. On lifting up the kidney after cutting through the pedicle a large tubular structure was found running up to the hilum. This complicated matters considerably as we were uncertain what it was. At first it was thought that it might be a large abnormal vessel. The only clue to its identity was that it lay in the usual position of the ureter. We knew, however, from the retrograde pyelogram that the ureter should be of normal size. Owing to the poor condition of the patient no time could be spent in isolating the various structures and the whole bundle was clamped and divided.

On removing the kidney it was found to have a double pelvis. The large duct which had worried us was in fact a grossly dilated ureter draining the upper pelvis. The lower ureter was adherent to it and completely obscured by it.

The wound was rapidly closed, leaving the clamp on the pedicle.

PROGRESS.—Post-operative progress was at first remarkably good. The clamp was loosened on the fourth day, and as there was no sign of hæmorrhage it was removed. This

was followed by brisk venous bleeding, which was controlled by packing. During the next few days there was some oozing which gradually stopped. On the eleventh day the pack was changed. This was again followed by a brisk hæmorrhage, mostly internal, which could not be controlled and the patient died.

POST-MORTEM EXAMINATION (Professor H. Haswell Wilson).—The site of the renal tumour was filled with a large blood-clot. The periphery of this was organized, but the centre was fluid. It connected directly with the inferior vena cava through a mass of necrotic growth round the renal pedicle. Death had evidently been due to a second hæmorrhage, through necrotic growth, into a partly organized hæmatoma. Secondary deposits were found in the left kidney, left suprarenal, and spleen. The lungs were covered by a delicate network of lymphatics permeated by growth. It was not possible to examine the bones.

The Kidney, Ureters, and Bladder.—The kidney is shown in *Fig.* 200. The upper ureter is greatly dilated, being about 1 in. in diameter. It drains what is left of the hydronephrotic pelvis and calices of the upper part of the kidney. It contains blood-clot. The lower ureter is of approximately normal size, and drains a greatly distorted lower pelvis. The tumour appears to have arisen from the lower part of the upper half of the kidney. In this region there is direct invasion of the pelvis. It has also involved the major portion of the remainder of the kidney apart from the lower pole. There is, however, a surprising amount of relatively normal tissue in the upper pole. The tumour was a pinkish-white colour and contained many large hæmorrhages. The outside of the kidney, though nodular, has not obviously been penetrated by growth. The perirenal tissues were, however, very adherent.

FIG. 200.—Hemisection of kidney, showing dilated upper ureter and pelvis containing blood-clot.

Microscopical examination of the tumour showed it to be a Grawitz carcinoma. There is considerable diversity in the arrangement of the cells, but the type (in the areas cut) remains remarkably constant. The cells are large, with finely granular cytoplasm, a big nucleus, and darkly staining nucleolus. Scattered throughout there are areas of hæmorrhage and necrosis. There is a well-marked fibrous stroma. Sections cut at the hilum show extensive infiltration of the perirenal fat by carcinoma. Tumour cells are also to be found in the lymphatics and veins of this region.

The two ureters descend to the pelvis together. In the abdomen the ureter from the lower pelvis lies behind the other and is obscured by it, but as they descend to the pelvis they rotate through 180° and the lower ureter comes to lie anteriorly. The vas hooks round both ureters in the normal situation. The lower (normal) ureter enters the bladder in the usual position. The ureter from the upper pelvis is grossly dilated and thinned throughout. It is more than an inch in circumference and admits the little finger easily. It passes through the bladder wall obliquely about $1\frac{1}{4}$ in. lower down (*Fig.* 201). The hypertrophied band of circular muscle-fibres which normally form the vesical sphincter are spread out as a broad cuff over its posterior surface. Indeed the ureter appears to pass right through the vesical sphincter to open into the extreme upper part of the prostatic urethra. The actual orifice is widely patent and admitted a 3-mm. rod quite easily. It lies above a normal prostatic colliculus. It would appear that the vesical sphincter controlled the orifice of the ectopic ureter at the same time as the bladder. This would explain the complete absence of symptoms, and also why there is still a considerable amount of functional renal tissue in the corresponding kidney. The grossly dilated ureter would act as an accessory bladder, and since there is no obstruction to the escape of urine during micturition back-pressure on the

kidney would be minimal. It must be remembered that the condition is congenital, and has, as it were, grown up with the kidney.

Comment.—The present case has been reported as it is a museum of abnormalities. The presence of a carcinoma in a kidney already the seat of so many other abnormalities is probably fortuitous. The interesting features are the enormous size of the supernumerary ureter, and the presence of a surprising amount of apparently functional renal tissue in this kidney. Although ectopic ureters commonly open into the prostatic urethra, they rarely do so at such a high level. Nearly all reports emphasize the high incidence of stenosis in ectopic openings. In this case the reverse was found, the orifice being considerably larger than normal.

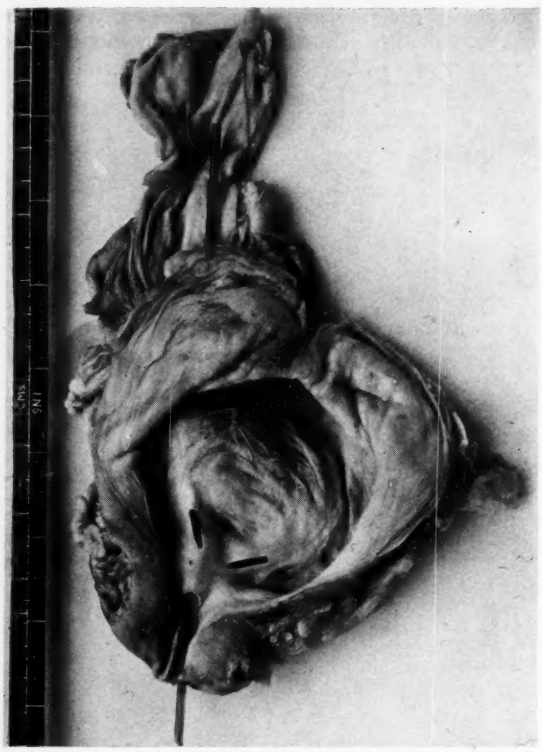

FIG. 201.—Bladder opened anteriorly. Glass rods in normal ureters (above), and ectopic ureter (below). Owing to contraction during fixation, the ectopic opening appears to lie rather higher than in life.

I wish to thank Mr. Bernard Ward for permission to publish this case, which was under his care, and also for much helpful advice. I am also indebted to Dr. H. Black for the pyelograms, and to Professor Haswell Wilson, who carried out the post-mortem examination. The preparation of all the specimens, sections, and photographs are the excellent work of Mr. W. H. O'Brien, technician to the Department of Surgery, Birmingham United Hospital.

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RESECTION OF GROWTHS OF THE LOWER PELVIC COLON: A NEW METHOD OF ANASTOMOSIS*

By SETON PRINGLE, Dublin

There has been of recent years a considerable revival of interest in the subject of the conservation of the sphincters when dealing with growths of the lower colon and rectum. In the early days of bowel surgery many different procedures were advocated, but the greatly increased risks which accompanied attempts to restore continuity of the bowel by any of the methods described led to their being discarded. Like many others, I have long felt that the condemning of all patients with lower pelvic colon growths to the discomfort of an abdominal anus was a reproach to surgery, but I considered that none of the methods so far advocated for conserving the sphincters so eliminated the dangers of sepsis as to be justifiable as routine procedures. It is for this reason that I now wish to report a method which I have employed and which may almost be described as 'aseptic'. It is, as will be seen from the drawings, founded on Coffey's third method for transplanting the ureters into the rectum.

OPERATION TECHNIQUE

The steps of the operation for a growth already located by a barium enema in the lower pelvic colon are as follows:—

First Stage.—The abdomen is explored through a small incision in the upper left rectus; the exact situation of the growth, its mobility, its general operability, the degree of lymphatic involvement, and the absence of metastasis in the peritoneum or liver, are all determined. Particular attention is paid to the length of the loop of the sigmoid colon and to the amount of bowel available below the growth. If my procedure is to be carried out a long sigmoid loop is necessary and the growth must lie at least four inches above the pelvirectal junction. If the case be considered suitable, a left transverse colotomy is performed in such a way that a good spur is left, completely diverting the fæcal flow as recommended by Devine, although I do not think the formation of a bridge of skin as described by him is necessary. During the following two or three weeks the lower bowel segment is frequently douched through from colotomy to anus.

Second Stage.—

Step 1.—The abdomen is reopened by a long left paramedian incision from umbilicus to pubis. The pelvic and if necessary the iliac and descending colons are mobilized so that after excision of the growth an ample length of the bowel will be available to reach the bottom of Douglas's pouch. The points for section of the bowel at least four inches above and below the growth are determined. The lower point of section should lie at the lowest at the pelvirectal junction, but if an inch or so of lower pelvic colon can safely be preserved the subsequent steps are

* Read before the Surgical Section of the Royal Academy of Medicine in Ireland, Feb. 16, 1940.

simplified. The mesentery of the segment of bowel to be removed is dealt with, and in doing so the superior hæmorrhoidal artery is preserved if possible. Should there be marked glandular involvement and a wide resection of the bowel and mesentery be necessary, the artery may be ligatured. In such a case it will soon be apparent whether or not a satisfactory circulation is being maintained in the rectal wall from the middle and inferior hæmorrhoidal arteries. If there is any doubt it is better not to employ the following method of anastomosis, but to deal with the condition by one or other of the methods entailing a permanent colotomy.

Step 2.—The bowel at a selected point above the growth is crushed with a very broad clamp. The extreme margin of the proximal bowel where it joins the upper limit of the crushed portion is encircled by a No. 6 silk suture on a needle which picks up the bowel at six or eight points (*Fig.* 202). This ligature is tightly tied, and one end left two or three feet long: to this is attached a small bull-dog forceps

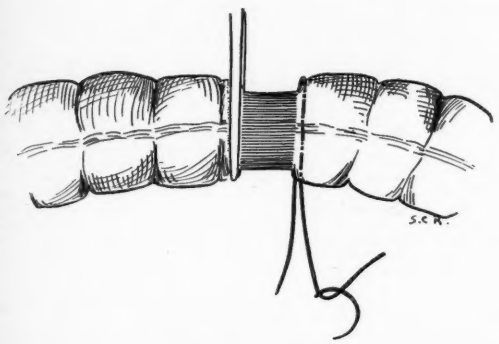

FIG. 202.—The method of dividing the bowel proximal to the growth. One end of the controlling suture when tightened is left long and attached to bull-dog forceps.

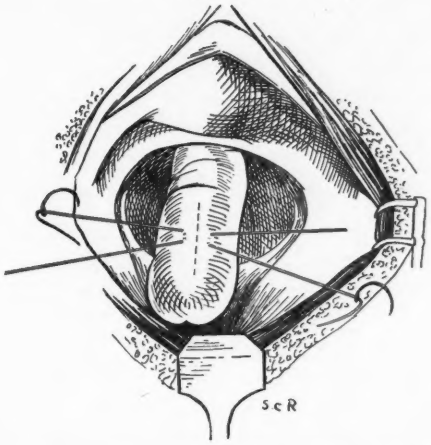

FIG. 203.—The rectal stump plugged with gauze is opened in the midline between two fixation sutures.

fitted with a spike. The lower part of the crushed segment is controlled by a light clamp and the bowel divided close to the silk ligature, the upper end being touched with liquid carbolic acid and the lower wrapped in a sponge wrung out of antiseptic. The bowel at the selected point below the growth is similarly crushed and divided, but here the ligature controlling the lower end is cut short and the stump invaginated.

Step 3.—The assistant now passes into the rectum the gauze insertor made by Collin of Paris. The upper end of the instrument is guided by the surgeon's hand to the upper blind end of the rectum. The entire rectum is rapidly filled with gauze wrung out of flavine solution. It is remarkable how accessible to the surgeon this procedure makes the anterior peritoneum-covered portion of the upper rectum. The rectal wall is picked up by two fixation sutures placed transversely and about a quarter of an inch apart (*Fig.* 203). These sutures are left long, with the needles still attached, and are held up by an assistant, thus making the bowel wall still more accessible. A 2-in. longitudinal incision is now made between the sutures through the entire thickness of the rectal wall on to the gauze. The bull-dog clip on the ligature controlling the proximal bowel picks up the gauze presenting in the incision (*Fig.* 204), and then an assistant withdraws the gauze and with it the ligature on the

upper bowel, so that the end of the latter is drawn down, guided by the surgeon, until it projects well into the lumen of the rectum (*Fig.* 205). It is there fixed by six or eight Lembert sutures snugly closing the rectal incision around it, the two fixation sutures being utilized for this purpose. The omentum is if possible drawn

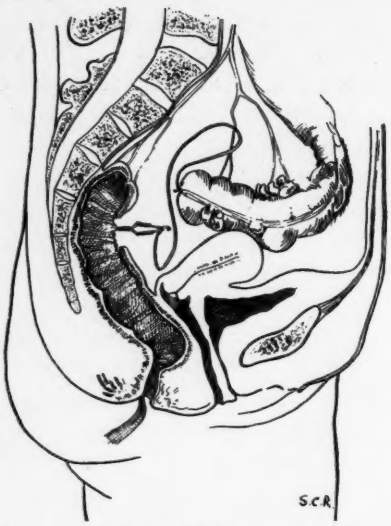

FIG. 204.—The gauze in the rectum is picked up by the bull-dog forceps.

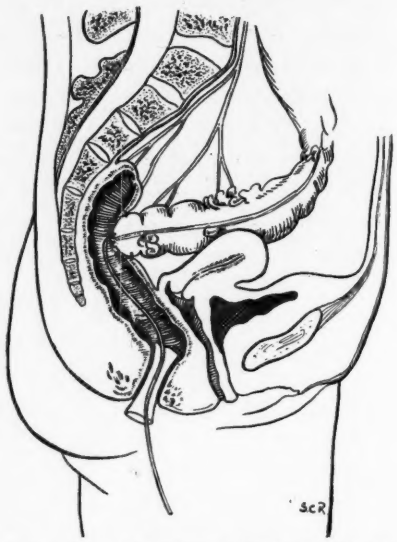

FIG. 205.—The gauze bringing with it the long suture on the end of the upper segment has been withdrawn and the closed end of the bowel invaginated into the rectum. A large tube occupies the anal canal.

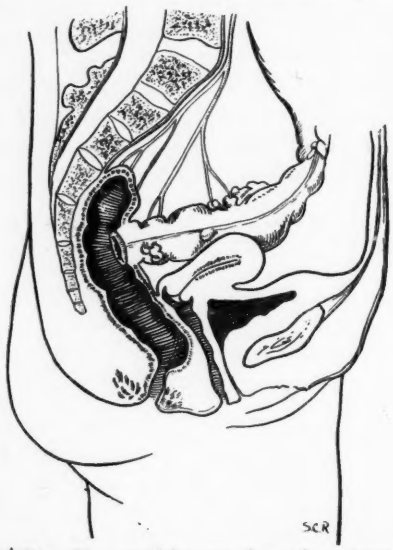

FIG. 206.—The condition ten days after operation.

down and sutured to the pelvic floor in such a way as to protect the small intestine from the raw surface, and the abdomen closed. The sphincter ani is stretched and a large-bore tube inserted through the anal canal.

Third Stage.—The ligature controlling the invaginated colon sloughs through in a week or so, thus reopening the bowel lumen (*Fig.* 206). In about a fortnight's

time an enterotome is applied to the spur on the colotomy, and as soon as this has been cut through the colotomy is closed and continuity of the bowel restored.

Instead of this three-stage method I have in some cases in which there had been no previous obstruction carried out the procedure in one stage, i.e., the preliminary colotomy was omitted and at the end of the operation—carried through as described for the second stage above—a cæcostomy was performed, thus precluding any great accumulation of fæces above or greatly increased tension on the invagination anastomosis. In these cases two ounces of liquid paraffin was daily injected into the cæcum, with the result that spontaneous bowel action occurred six or eight days later—in other words, shortly after the controlling ligature had cut through and the continuity of the lower bowel lumen become restored. The cæcostomy as a rule closed spontaneously, but if not, its closure under local anæsthetic was but a minor procedure.

DISCUSSION

I would suggest that this operation presents very definite advantages: (1) It enables the anastomosis to be carried out deep in the pelvis without any appreciable risk of sepsis. The mucous membrane of neither colon nor rectum is handled even with a forceps. No sutures penetrating the mucous membrane are employed, and there is no fæcal flow over the line of anastomosis until healing is well advanced. (2) It enables certain growths in the lower pelvic colon to be satisfactorily dealt with without sacrificing the sphincters. (3) The major stage (*Stage* 2) can be rapidly performed, requiring rather less than an hour.

ILLUSTRATIVE CASES

I append a note of only two cases, as each taught me a useful lesson. *Case* 1 was an example of the full three-stage operation in which the special technique described above worked smoothly and well, but which unfortunately ended fatally from an accident during the closure of the colotomy. *Case* 2 is an example of a successful result of the method modified to a one-stage operation.

Case 1.—Mrs. M. S., aged 59, admitted to Drumcondra Hospital on May 25, 1938. Two years' history of blood in stools; three months persistent diarrhœa accompanied by slight colicky pains.

On Examination.—Abdomen negative; growth just palpable by tip of finger per anum; sigmoidoscopic examination confirmed the presence of a malignant stricture of lower pelvic colon.

Treatment.—

May 28, 1938: Exploratory laparotomy—movable growth in lower pelvic colon immediately below promontory of sacrum, peritoneal coat not involved, local glandular involvement, no palpable deposits in liver. Left transverse colotomy, afferent and efferent loops being sutured together. Lower segment washed through to anus daily until June 9.

June 9: Abdomen reopened by a long paramedian subumbilical incision; iliac and pelvic colons mobilized; superior hæmorrhoidal artery divided at origin; growth resected as described above; rectum plugged with gauze, making the anterior peritoneal-covered wall easily accessible; end-to-side anastomosis as described completed without difficulty. Convalescence was normal, the silk ligature controlling the lower end of the upper segment coming away on the seventh day. Excellent union of colon to rectum took place, the opening being easily palpable by a finger in the rectum.

June 17: A Devine enterotome was applied to the spur. The patient immediately complained of some pain with nausea, which unfortunately I did not regard seriously, but two days later the tightening up of the enterotome was followed by grave shock, collapse, and vomiting.

The enterotome was loosened after several hours and I hoped all would be well, but general peritonitis developed and ended fatally six days later.

POST-MORTEM EXAMINATION.—It was difficult to distinguish the exact cause of the peritonitis, but apparently I had not made a sufficiently long spur when performing the colotomy; a loop of jejunum had become adherent to the base of the spur, and had been nipped in the long blades of the Devine enterotome.

Case 2.—Mrs. D., aged 34, admitted to the Royal City of Dublin Hospital on March 15, 1938, under the care of my gynæcological colleague, Dr. Ninian Falkiner. In the course of an operation on March 24, Dr. Falkiner found an early carcinoma of the lower pelvic colon situated opposite the sacral promontory and encircling less than half the circumference of the bowel. He kindly sent for me as I happened to be available.

On examining the condition I decided to do a one-stage operation as the patient had been carefully prepared beforehand and as there was no evidence of obstruction. I accordingly divided the superior hæmorrhoidal artery, stripped down the mesentery from the anterior surface of the sacrum and carried out the resection and anastomosis as described without difficulty, concluding the operation by performing a cæcostomy.

The convalescence of this patient was completely normal. Paraffin was given daily by the mouth and two ounces injected into the cæcostomy every second day, with the result that the controlling silk ligature came away on the sixth day and a spontaneous bowel evacuation took place that evening. The cæcostomy healed spontaneously and she was discharged from hospital in excellent condition on the twenty-sixth day after operation.

She presented herself for examination on Feb. 7, 1940. In the interval the bowel had been acting normally and regularly, the only aperient required being an occasional dose of liquid paraffin. Her general condition was excellent. The anastomosis, which could be easily palpated by the finger in the rectum, was smooth and there was no evidence of induration in the surrounding bowel. There was no evidence of local or general recurrence.

My thanks are due to Mrs. Stella Henry for the drawings which accompany this paper.

EMPHYSEMA OF THE BILIARY PASSAGES

By P. H. WHITAKER
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AND JOHN P. STEEL
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DELINEATION of the biliary passages by the presence of gas when seen in a radiograph is a sufficiently rare condition to warrant the description of a case in which the whole of the biliary passages were outlined in this way.

Normally the ampulla of Vater is a sufficiently powerful sphincter to prevent regurgitation of food or fluid from the duodenum entering the bile-passages. Cases are recorded, however, where it has been possible in pathological conditions to force opaque materials into the ducts in this way.

A case of patency of the ampulla was recorded by W. E. Pennington[1] in 1932. In this instance there was present a carcinoma of the pancreas which was causing partial obstruction of the duodenum. The growth had smoothed out the rugæ of the duodenum and infiltrated the ampulla so that a barium fluid could be palpated into the whole of the bile-tract and demonstrated in this way.

Otto Wichtl[2] also recorded a case in which the barium meal entered the passages, which were, however, poorly filled.

In neither of the above instances, however, was air discovered in the liver.

Hans Heinrich Berg[3] found that careful examination of the whole liver made under the optimum conditions of exposure revealed with surprising frequency signs of gas filling the biliary passages. He ascribes the causes as being due to spontaneous biliary fistula, insufficiency of the ampulla of Vater due to the passage of stones, and infection of the biliary tract with gas-forming organisms. Perforations may take place in connexion with the colon, when it may be demonstrated by an opaque enema, and into the duodenum in the cases of perforation of a duodenal ulcer. If the formation of gas takes place in the closed biliary passages they are more clearly demonstrated.

Ernst A. Schmidt[4] also records two cases of emphysematous cholecystitis both of which showed the presence of gas in the gall-bladder, and this was confirmed by the finding of an identical shadow after cholecystography. In his cases the tracts, apart from the gall-bladder, were not visible.

It would appear, therefore, that the presence of gas in the biliary passages can be accounted for in the following ways: (1) Infection with gas-forming organisms; (2) Perforation of the gall-bladder in connexion with the colon or small bowel; (3) Perforation of a duodenal ulcer into the common bile-duct; (4) Interference with the ampulla sphincter by cicatrization, growth, or the presence of a stone.

CASE REPORT

HISTORY.—A woman, aged 57, on admission to hospital gave a history of epigastric pain travelling through to the back, unrelated to food but accompanied by vomiting; she had been ill at home for a week, but had gone to bed because of the sudden onset of pain

"in the stomach" whilst she was out shopping. She said she had had a similar attack some six months ago, when the sequence was the same, and which she described as pain across the stomach relieved by vomiting which was very white and frothy; flatus and constipation were marked.

She was admitted to another hospital, which she left against advice before a provisional diagnosis of pancreatitis could be investigated, and after this her diet was practically free from fat, eggs, or potatoes, all of which caused her discomfort.

Four years ago she had another attack, with pain over the chest and penetrating to the back, breathlessness and vomiting, and lasting twenty-four hours at least.

She remembered that many years ago she had suffered from jaundice, and since the acute attack in 1933 she had lost weight and deteriorated in her general physical condition.

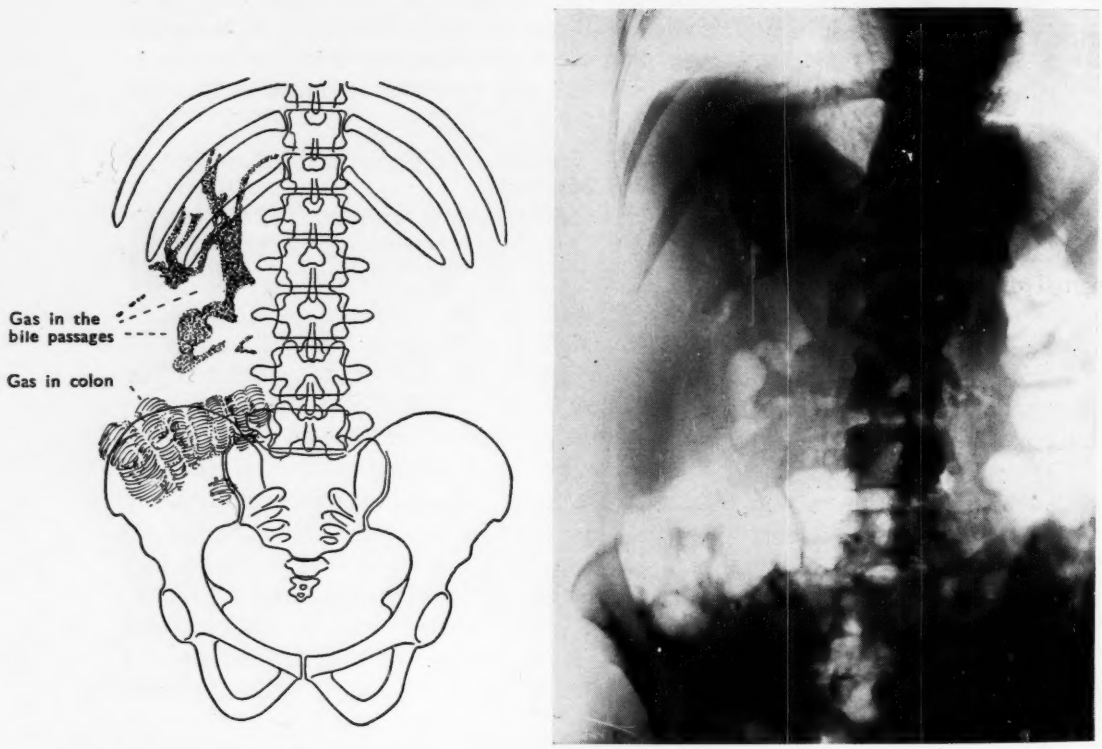

FIG. 207.—Radiograph, with key diagram, showing gas-distended gall-bladder and biliary passages.

ON EXAMINATION.—On admission to this hospital in March there was a slight degree of jaundice, considerable guarding in the right hypochondrium, and pain in the back, with some frequency and scalding on micturition. The liver was enlarged three finger-breadths downwards, and tenderness was present over the whole margin. The heart was slow; there was considerable gaseous distension of the abdomen, but no sign of free fluid. The skin was loose, with slight pitting on pressure over the abdomen.

The jaundice persisted and stools were clay coloured, urobilin being found in the urine.

The Wassermann reaction was negative; the blood-count showed 3,000,000 red cells and 10,000 white cells, a moderate leucocytosis, and an absolute increase of polymorphs to 90 per cent.

The Van den Bergh test was negative, the N.P.N. 49 mg. per cent, the urinary diastase 6·6 units, the icterus index of the blood 17, and the blood-culture remained sterile for three days, whilst the electrocardiogram showed no abnormal features.

Radiographs were taken after the administration of opacol. In three there was no evidence of any of the dye concentrated by the gall-bladder, although dye was seen in the bowel. In the liver region, the margin of which can be clearly seen, there was a dense shadow of the gas-distended passages. The main branches are dilated and show ramifications throughout the liver substance. The cystic duct is also dilated, but the gall-bladder, which is clearly seen, is of normal size (*Fig.* 207). Radiographs were taken at three intervals during twenty-four hours and the findings were constant, but on examination the following day all traces of the air had vanished. The striking feature of this finding was that there was no sign of any calcified stone shadow, but at the lower end of the common duct there was a rounded shadow, the upper margin of which was outlined by gas shadows; this was of the density of the normal tissue, but was later found to be due to a soft impacted stone.

The patient died.

POST-MORTEM EXAMINATION.—There was a large abscess localized by the upper surface of the liver, the anterior abdominal wall, and below and behind by adhesions and the pyloric end of the stomach and duodenum. On careful dissection it was possible to free the pylorus, which was bound down by adhesions, and to open the duodenum from the inside. The duodenum was patent throughout its whole course and there was no evidence of any abscess or perforation. The ampulla was projecting into the lumen of the duodenum, which was widely dilated by a round soft substance which proved to be a large soft gall-stone, very friable and about half an inch in diameter. This was protruding into the lumen of the duodenum. The bile-passages were dilated and inflamed, as also was the gall-bladder, and the liver round the hilum showed several small metastatic abscesses. There was no evidence of any further gall-stone. No gas-forming organisms were found in the pus, nor obtained from the passages.

Comment.—We think that it may be assumed that the stone, until it became impacted, had acted as a ball valve in the ampulla and allowed gas to enter from the duodenum. Impaction had occurred immediately following the cholecystograph, and the air imprisoned in the biliary tract had been absorbed before the second examination.

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SHORT NOTES OF RARE OR OBSCURE CASES

AN UNUSUAL CAUSE OF INTESTINAL OBSTRUCTION

By J. J. M. SHAW
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ALTHOUGH an extensive literature exists on the subject of duodenal diverticula and their complications, I have been unable to trace a record of the condition now described. It consisted of the blockage of a distal part of the bowel by a large calculus extruded from a diverticulum of the duodenum.

In a consecutive series of 2000 barium examinations, Digby Wheeler[1] found 7·1 per cent of primary duodenal diverticula. This figure refers to the second and third parts of the segment; the first part is excluded, as in it the great majority of recesses are associated with old ulcer formation.

Harold Edwards,[2] in his admirable Jacksonian Prize Essay of 1932, describes a typical primary duodenal diverticulum as a thin-walled sac opening into the bowel by a wide mouth. Although other factors, and especially the nature of the contents, may have a bearing upon calculus formation, it is obvious that the relationship between the size of the cavity and the width of its outlet must be of major importance, as exemplified in the gall-bladder.

Many cases have been described in which collections of food and epithelial debris or actual calculus formation have given rise to symptoms of irritation and infection. The comparative immobility of the duodenum and associated diverticula is sufficient to prevent the occurrence of the interesting complication which Charles M. Watson[3] described in relation to the effect of enterolith formation in a diverticulum of the jejunum. In his case, the weight and twisting action of the contents of the recess caused kinking and ultimate obstruction.

In the case now under review, the assumption is warranted that a large enterolith which was removed from the middle third of the ileum had been formed in and extruded from a large diverticulum of the duodenum which was demonstrated radiographically. The barium was administered in a subacute phase of the obstructive attack and one photograph shows the diverticulum in a state of spasm.

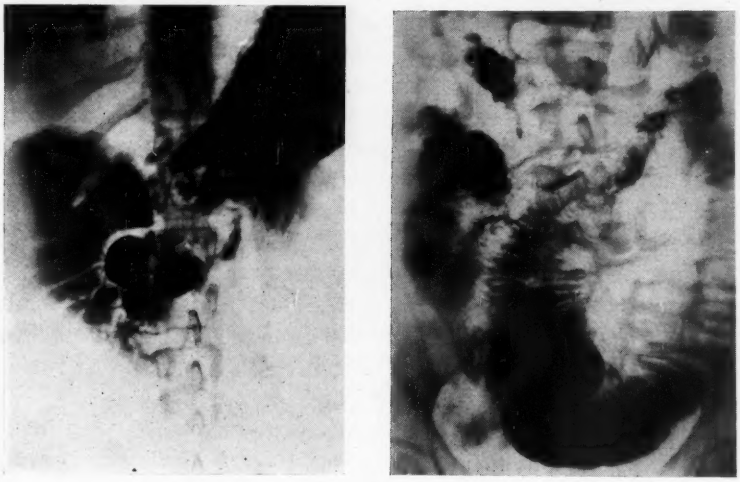
CASE REPORT

Female, unmarried, aged 56 years. General health good. Gave history of five sudden attacks of vomiting without obvious cause at long intervals. First attack in 1922, and others of sufficient severity to have been specially noted by patient.

April 18, 1938: Sudden onset of vomiting, but on the third day, instead of recovery as previously, she felt ill and was unable to rise from bed. Bowels failed to move for seven days in spite of enemata.

April 25: Transferred to Edinburgh, when bowels moved freely after high olive-oil enema. Vomiting ceased for three days, and a barium series was taken. This showed small-bowel obstruction (*Figs.* 208, 209), but the cause was not obvious

as a large duodenal diverticulum was thought to be merely a coincident finding and unrelated to the obstructive factor. Vomiting restarted on the fifth day after



FIGS. 208, 209.—Radiographs after barium meal showing the duodenal diverticulum.

admission, with a huge residue in the stomach on lavage. The general condition had remained satisfactory throughout and the cause was obviously mechanical and intermittent.

Laparotomy was carried out on the sixth day, when an enterolith was removed from the ileum. The patient made a good recovery and left the nursing home in the fourth week. X-ray examination, six months later, showed no lesion in the biliary tract..

The stone measured 4·5 cm. by 3 cm. (*Fig.* 210). Its chemical analysis was "mainly lipoid material with traces of cholesterol, uric acid, phospho-lipins, calcium soaps, and bile-pigment".

The size and shape of the stone, its chemical composition, and the radiological evidence all support the view that it had developed in the duodenal diverticulum.

FIG. 210.—Enterolith removed at operation. (*Natural size.*)

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RARE CONGENITAL MALFORMATION OF THE UPPER LIP

BY WALTER RADCLIFFE, WIVENHOE

A. B., a girl patient aged 6 years, was brought to me by her mother on account of mild catarrhal otitis media. During examination of the mouth two small nodules were noticed on the upper lip. They are approximately hemispherical, about 1 mm.

in diameter, situated symmetrically a very little way from the margin of the mucous membrane, and about half way between the centre of the lip and the corner of the mouth on either side. The colour is a shade darker than the surrounding mucous membrane, and more especially on the left side the centre appears to be slightly bluish and transparent, and the whole nodule looks cystic; this cystic appearance is not so marked in the right-hand nodule.

The child's mother told me that they had been noticed soon after birth, and that they caused no inconvenience, and for the last year or two they do not appear to have grown any larger. The parents do not wish to have them removed, so that microscopical examination is not possible.

I take them to be small inclusion cysts formed during the fusion of the embryonic lip elements. Sir Arthur Keith informs me that they are doubtless aberrations in the development of the lip, but that they do not represent any structure in the lower vertebrates. Crypts in the lower lip in situations corresponding to these nodules sometimes occur (Brophy), but I have been able to find only two references to similar structures in the upper lip (Ludy and Shirazy), both cases unilateral, and taking the form of minute crypts or pits in the mucous membrane.

I am indebted to Sir Arthur Keith for pointing out that the photograph (*Fig.* 211) clearly shows imperfect fusion of the upper lip, most marked on the right side. The fleshy ridge corresponding to the globular process, and running down from the nostril towards the nodule on that side, is quite clearly shown (*see also Figs.* 212, 213), and a little pit in the centre of the lip marks the interglobular space (A). The groove between the globular process and the lateral nasal process (C) on the right side is also clearly shown. On the left side the fusion is more complete and is nearly normal.

FIG. 211.—Photograph of upper lip showing the cysts. Imperfect fusion is most marked on the right side.

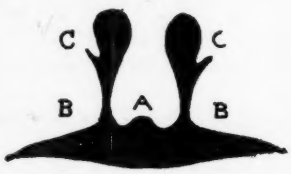

FIG. 212.—Embryonic lip elements. A, Inter-globular space of the mesial nasal process; B, Maxillary processes; C, Lateral nasal processes.

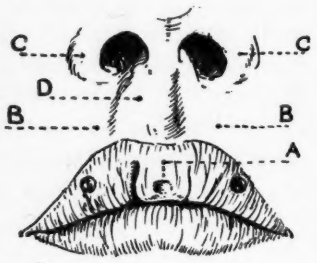

FIG. 213.—Drawing from the photograph. The significance of the lettering is the same as in *Fig.* 212. D, Ridge corresponding to the right globular process.

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REVIEWS AND NOTICES OF BOOKS

Berkeley Moynihan, Surgeon. By DONALD BATEMAN. With a Preface by Lord Moynihan. 8½ × 5½ in. Pp. 355 + xv, with 8 illustrations. 1940. London: Macmillan & Co., Ltd. 12s. 6d. net.

THIS biography of a great man has been eagerly awaited by his many friends and admirers. And now that we have it we feel it to be a work of outstanding merit. It is very accurate in its details, which have been supplied by members of his family. It is written by a college friend of his son who has shown great skill in depicting the life of his hero through its various phases. The author, being a medical man practising in the North of England, is conversant with the medical problems, both scientific and ethical, which concerned Moynihan's career, but for the purpose of enlightening non-medical readers he gives an intelligible account of the technical subjects concerned. The most fascinating part of the book is the account of Moynihan's early life and the tragedies which beset his parents. His father was a Serjeant in the Army and one of the first to win the V.C., which he did in the Crimean War. But Captain Moynihan only lived to be 37 when he died of undulant fever at Malta. His mother's struggle for existence, with three children to bring up on a pound a week, gives us some appreciation of Moynihan's merit in gaining his education and leading position in his profession.

In the description of his career free use is made of quotations from his speeches and writings, which serve to illustrate his eloquence and enthusiasm, and it is stimulating in these dark days to read what he said about the eternal fight between tyranny and freedom. It is of course impossible for any biography to be written so soon after a man's death, and in the lifetime of his children, which can, be anything but eulogistic. Dr. Bateman has, however, fairly admitted, although he has also excused, the frailties of his subject, but a generation or so must pass before a fair assessment can be given of the part which Moynihan played in the building up of modern British surgery. In the meantime we have a delightfully written story of one who played a great part in the surgical life both of England and America.

Rose and Carless' Manual of Surgery. For Students and Practitioners. By CECIL P. G. WAKELEY, D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S., F.R.A.C.S., Fellow of King's College; Senior Surgeon, King's College Hospital; etc.; and JOHN B. HUNTER, *M.C.*, M.Chir., (Cantab.), F.R.C.S. (Eng.), Surgeon, King's College Hospital; etc. In two volumes. Sixteenth edition. 9¼ × 6¼ in. Pp. 1756 + xii, with 1090 illustrations and 19 coloured plates. 1940. London: Baillière, Tindall & Co. 30s. net.

IT is a real pleasure to anyone whose earliest surgical reading was founded on this text-book to find how it has grown from strength to strength and worthily maintained the traditions of Lister as well as those of its original authors.

The present edition appears to contain a great deal that is new both in text and illustrations, but by a system of judicious elision and compression it is only actually 90 pages longer than the last edition; yet it has 300 new illustrations, most of them by that incomparable artist, Maxwell. The earlier pathological chapters have been somewhat compressed, this affecting chiefly the sections on bacteriology and hæmatology, hæmorrhage and shock, ulceration and gangrene. Quite rightly war surgery is chiefly dealt with as a part of traumatic surgery, and the small special chapter on the subject seems hardly necessary. It must be extremely difficult for the editors of a book like this to avoid giving undue prominence to the speciality in which they themselves are interested, and we notice in the present work that the surgery of the central nervous system has been dealt with in great detail both in the text and the illustrations. We are inclined to question whether it is wise to represent to students and practitioners such rare conditions and operations as the intramedullary tractotomy of the fifth nerve, the opening of the spinal cord for syringomyelia, the cauterization of the choroid plexuses for hydrocephalus, pineal tumours, or focal epilepsy.

Some of the old illustrations, such as the ligature operation for aneurysm, might well be omitted and those of the more modern operations of Matas might be included. Hodgkin's disease is described without mentioning the tendency to bone metastasis. The arrangement of the order of some of the chapters is curious, those on the ear and the eye being placed at the end, after amputations and gynæcology. The sections on deformities and fractures seem to be rather on a lower level than those on the brain and the abdomen, and we think that the treatment of fractured patella by removal of the fragments, and the subject of hindquarter amputation, deserve mention.

The book as a whole represents a most complete and well-illustrated presentation of modern surgery, and we are surprised that the publishers can afford to give such good value for the money.

Fractures and Dislocations for Practitioners. By EDWIN O. GECKELER, M.D., F.A.C.S. Second edition. $9\frac{1}{8} \times 6\frac{1}{8}$ in. Pp. 314 + xii, with 267 illustrations. 1940. London: Bailliere, Tindall & Cox. 22s. net.

THE object of this book is the laudable one of giving the essentials about fractures and dislocations for practical purposes.

We think it is a pity that the official status of the author is not given, and we rather infer from this that he must be a tutor or lecturer. It is, of course, very difficult to write a small book giving all the essentials and omitting all the unnecessary details in an important subject. To be of any practical use the details of each method must be given sufficiently clearly to enable the reader to carry it out. This test is often not satisfied. For example, fractures of the patella are dealt with in four pages and the details necessary to carry out the treatment are so meagre as to need reference to another book. On the other hand, many of the illustrations, e.g., those describing remedial exercises, are quite unnecessary. The illustrations are numerous and good. The letterpress is very readable. In the matter of treatment, chief reliance is placed on the use of plaster-of-Paris, the Thomas splint, and skeletal traction. We should have thought that Cramer's splinting would have been included among the list of appliances to be kept in stock.

Pye's Surgical Handicraft. Edited by HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital, London; Surgeon and Urologist, Essex County Council; External Examiner in Surgery, University of Bristol; etc. Twelfth edition, fully revised. $8\frac{1}{2} \times 5\frac{1}{2}$ in. Pp. 595 + xii, with Frontispiece and 482 illustrations, some coloured. 1940. Bristol: John Wright & Sons Ltd. 21s. net.

QUITE certainly Walter Pye (1853–92) would not recognize in this large, handsome volume his modest *Surgical Handicraft*, which he first published in 1884.

Now comes the twelfth edition, which contains almost everything in the way of technique a House Surgeon might want to know. It is a book to have and to keep handy for reference.

Operative Surgery. By J. SHELTON HORSLEY, M.D., LL.D., F.A.C.S., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va., and ISAAC A. BIGGER, M.D., Professor of Surgery, Medical College of Virginia; Surgeon-in-Chief, Medical College of Virginia Hospitals, Richmond, Va. With contributions by C. C. COLEMAN, M.D., F.A.C.S., AUSTIN I. DODSON, M.D., F.A.C.S., JOHN S. HORSLEY, jun., M.D., and DONALD N. FAULKNER, M.D. In two volumes. Fifth edition. $10 \times 6\frac{5}{8}$ in. Pp. 1567 + xxvi, with 1391 illustrations. 1940. London: Henry Kimpton. 90s. net.

THE popularity of this book is indicated by the fact that this, the fifth, edition has been called for only three years after the last. The authors are the same, and thus have the advantage of their previous experience and the pooling of the criticisms they have received in the meantime.

In spite of the difficulties, the balance of the work has been maintained, obsolete material having been deleted and new procedures incorporated; many of the latter appear to have been quoted merely to keep the work up to date and not as having a very firm basis for their adoption; even the authors themselves do not appear to be very convinced of their efficacy, e.g., "the intravenous alimentation of proteins by solutions of amino acids" as recommended by Elman and Weiner, and the administration of intravenous saline with alcohol (Mueller's solution). The chapters on the principles and technique underlying the treatment of malignant disease are critical and stimulating. "The manner in which a person dies is often more

impressive than the actual death". "Big accidents are front page news . . . but the fact that 140,000 persons die yearly of cancer in U.S.A. makes little impression". The use of radium and deep X-ray therapy might be given more prominence in this section. If these adjuncts to surgery were recommended more, there would not need to be so many plastic procedures described in subsequent portions of the work. The formation of a 'blood bank' is urged, but the Lindenmann syringe method recommended for transfusion seems clumsy and fussy.

A chapter is devoted to "reversal of the circulation" in the treatment of incipient gangrene of the lower extremity, which ends with the encouraging conclusion that "the same result can be obtained much more accurately and with less danger simply by ligation of the femoral vein under a local anæsthetic". The operative procedures for dealing with fractures are fully considered, but the importance of rest and plaster extension is not stressed as it should be; if it were, then many of the operations described would not be necessary. Excision of the patella is mentioned, but obviously the author has had no experience in this treatment.

Wound treatment is dealt with rather too briefly considering its outstanding importance; suture of muscles and fascia is recommended. Immediate skin-grafting in closing a defect in the skin is not mentioned, but various methods of sliding grafts are recommended. This may add considerably to the danger if the original wound suppurates.

Various orthopædic operations are included in the section on the operations for fractures. It would have been easier reading if the newer orthopædic procedures had had a special section given to them.

The section on thoracic surgery is extremely well done and beautifully illustrated.

In the section on herniorrhaphy silk is the suture material recommended, even in preference to autogenous fascia. The latter is said to be often absorbed, leading to recurrence (27·7 per cent), and also infection is more common. The injection treatment is condemned, the figures of Burdick and Coley being quoted, viz., 47 recurrences out of 56 cases so treated at the New York Hospital for Ruptured and Crippled.

In closing an abdominal incision, tension sutures are recommended to pierce all the layers of the abdominal wall, including the peritoneum. This seems to give little advantage and adds a distinct element of risk to the patient.

Suture of the wound in cases of appendiceal abscess is limited to the peritoneum only and then the rest packed with vaseline gauze.

In the section on the 'acute abdomen' the difficulty of excluding coronary thrombosis in certain cases is rightly emphasized; but few surgeons will agree that thrusting artery forceps through the mesocolon into the pancreas, and putting drainage tubes into the cavities thus made, has any sound pathological basis for the treatment of acute pancreatitis. The same criticism applies to the statement that while drainage is unnecessary in a perforated peptic ulcer, in the case of a perforation due to cancer it will prevent the implantation of cancer cells.

Startling statistics are given as to successful operations for acute appendicitis with or without abscess and peritonitis, the death-rate being 0·8 per cent. This is attributed to five factors: (1) Immediate operation *whatever* the stage of the disease; (2) McBurney's incision, the appendix is *always* removed; (3) Suction is employed instead of sponging or 'packing off'; (4) The stump of the appendix is *never* invaginated; (5) Post-operative physiological rest by limiting fluids orally and rectally (intravenous Ringer's solution if necessary is given and the duodenal tube employed). The method of dealing with the incision has already been mentioned.

The principle of physiological rest is stressed in operations on the stomach, or at least that is the reason given, together with the probability of acute dilatation, for urging a temporary gastrostomy in all cases requiring an interim gastric operation. The definite indications for this procedure are not given, nor is it urged after the suture of a perforated ulcer.

A Billroth II or some modification is preferred for gastrectomy; Shoemaker's method is not mentioned, nor excision of the omentum in gastric carcinoma.

The section on brain surgery is very full. Dandy's epoch-marking discovery in ventriculography in 1918 is credited with the opening of a new era in intracranial diagnosis.

The section on genito-urinary surgery is also very replete, though here again it is hard to believe that some of the procedures described are practised by the authors. The authors prefer perineal prostatectomy to the suprapubic route and obviously have had little experience with the McCarthy resectoscope or the Thompson-Bumpus punch.

The surgery of the rectum is sketchy and valuable space is taken up by a description of the Kraske technique as a one-stage operation. There is no general exploration of the abdominal cavity, and an incontinent sacral anus is established.

The work is well worth reading and will form a valuable book of reference. The format of the book is excellent and the illustrations very good. It would be a great addition to the handiness of the work if in future editions the publishers would put the excellent index in both volumes instead of only in the second.

Theory and Treatment of Fractures of the Jaws in Peace and War. By HORACE HAYMAN BOYLE, H.D.D. and L.D.S. (R.F.P.S. Glas.), H.D.D. (R.C.S. Edin.), late Captain, D, Army Medical Service; Dental Surgeon to the Ministry of Pensions; etc. 9¾ × 6 in. Pp. 288, with 127 illustrations. 1940. London: Henry Kimpton. 21s. net.

AS its title implies, this book is divided between theory and practice. The theoretical part deals with the curves of occlusion and does not seem to have very much practical bearing. For the most part the book consists of illustrations of interdental fixation by splints and wiring. Most of these are full size and very clear. The sections on plastic surgery are chiefly quotations from Kilner and Gillies, and are too short to be of any practical value. There is a description of kallomix, a new plastic substance, which is recommended for making dental appliances.

Cyclopropane Anæsthesia. By BENJAMIN HOWARD ROBBINS, B.A., M.S., M.D., Associate Professor of Pharmacology, Vanderbilt University School of Medicine. 9 × 6 in. Pp. 175 + xii, with 40 illustrations. 1940. Baltimore: Williams & Wilkins Co. (London: Baillière, Tindall & Cox.) 16s. 6d. net.

MOST of the volumes on the subject of anæsthesia which might find a place in an anæsthetist's library are of a comprehensive nature, and offer themselves for consideration because of their endeavour to include in their pages all types of anæsthesia. So outstanding a method as spinal anæsthesia soon asserted its individuality by the publication of books devoted entirely to this subject, and it was to be expected that ere long two other members of the growing anæsthetic family—intravenous anæsthesia and cyclopropane anæsthesia—would assert their independence. With regard to the latter, expectations have been fully realized by the appearance of this monograph by Dr. Robbins.

This book on a subject of keen interest to surgeons as well as anæsthetists, brings together the facts about cyclopropane, and embodies in one text the knowledge which has hitherto been scattered in a vast amount of literature. The bibliography at the end of the volume includes no less than 206 references, a round dozen of the original papers being published by the author and his co-workers. These papers, as well as the text, reflect the author's absorbing interest in quantitative analysis as well as in the effects of various methods of premedication, whereby the concentration of cyclopropane may be reduced and the safety margin increased.

Special attention is paid to the effect of cyclopropane on the circulatory and respiratory systems, and the author quotes many interesting observations on these aspects based on electrocardiographic and other observations, both in man and in experimental animals.

This volume will repay study by all interested in cyclopropane anæsthesia. It is clearly printed, pleasingly bound, and contains a multitude of explicit tables and clearly reproduced electrocardiographic and experimental records.

Cancer in Childhood. And a Discussion of Certain Benign Tumors. Edited by HAROLD W. DARGEON, M.D., F.A.A.P., Attending Pediatrician, Memorial Hospital for Cancer and Allied Diseases, New York; etc. 10 × 6½ in. Pp. 114, with numerous illustrations. 1940. London: Henry Kimpton. 15s. net.

THIS little monograph is written by eleven American surgeons and physicians, all of whom are associated with the Memorial Hospital for Cancer in New York. It is not quite clear what is meant by childhood. The growing period of life extends up to twenty-five years, but it is the first ten years of life that is chiefly considered in this book.

Neoplasms in children do not present any distinguishing features from similar growths found in adults, but the most interesting point of difference is their distribution and the frequency of special types. The tissues most frequently affected are the central nervous system, the bones, the eye, and the kidney. The frequency of sarcoma is much greater than that of carcinoma. No doubt many of the growths have a congenital origin. Some of the tumours which are relatively commoner in children than in adults are the following: Ewing's bone sarcoma, glioma of the eye, and Wilms' tumour of the kidney. The prognosis of most of these growths is very bad, and as regards treatment there is a general agreement that the best method is preliminary radiation, followed by excision or amputation.

The Pineal Organ. By REGINALD J. GLADSTONE, M.D., F.R.C.S., F.R.S.E., D.P.H., Formerly Reader in Embryology and Lecturer on Anatomy at King's College, University of London, and Lecturer on Embryology at the Medical College, Middlesex Hospital, London; and CECIL P. G. WAKELEY, D.Sc., F.R.C.S., F.R.S.E., F.Z.S., F.A.C.S., F.R.A.C.S., Fellow of King's College, London, and Lecturer in Applied Anatomy at King's College, University of London; Senior Surgeon, King's College Hospital and the West End Hospital for Nervous Diseases; etc. 10 × 6½ in. Pp. 528 + xvi, with 324 illustrations. 1940. London: Baillière, Tindall & Cox. 42s. net.

IN spite of recently acquired knowledge about the physiology and pathology of the pineal body, much remains unknown which, when it is finally revealed, is likely to prove invaluable to the clinician and especially to the surgeon who is called upon to diagnose and attempt to remove tumours of this organ. In the present volume the authors survey the comparative anatomy, one of the chief foundations upon which further investigations may be based, and they give brief accounts of the clinical picture and the operative treatment of pineal tumours.

The chapters on the types of vertebrate and invertebrate eyes, and on median and lateral eyes, though doubtless of much interest to the comparative anatomist, are unlikely to prove equally attractive to the surgeon because there seems to be no clear indication that this study gives him any help in solving his own problems. The sections dealing with the structure and development of the human pineal, its function and pathology, are more to the point, but their connexion with the pineal chapters on surgical technique still seems rather indefinite.

As a contribution to comparative anatomy, the work may be of real value; but as a guide to the clinician and especially to the surgeon it can scarcely be considered to have achieved its object.

A Handbook of Radiography. By JOHN A. ROSS, M.A. (Camb.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), D.M.R.E. (L'pool), Radiologist, Warrington Infirmary; etc. 8½ × 5½ in. Pp. 126 + viii, with 67 illustrations. 1940. London: H. K. Lewis 7s. 6d. net.

THIS little book is intended to be a kind of pocket handbook as a guide to the practice of radiology. It is illustrated by simple line drawings and is intended for the use of the students. It describes the ordinary methods and exposures for simple radiography, including the use of the various opaque media. There is also a short account of the more recent methods such as tomography, seriescopy, and kymography. It has a large number of blank sheets included in the text for the purpose of note taking.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Toracoplastias. Consideraciones clínico-quiriúgicas sobre las Toracoplastias en los Tuberculosos. By Dr. JORGE A. TAIANA. 10½ × 7 in. Pp. 98 + xxi, with 58 illustrations. 1939. Buenos Aires: El Ateneo.

Surgery of the Hand. By R. M. HANDFIELD JONES, M.C., M.S., F.R.C.S., Surgeon to Out-patients, St. Mary's Hospital; Senior Surgeon, Florence Nightingale Hospital; etc. 9¾ × 6⅞ in. Pp. 140 + viii, with 95 illustrations, several in colour. 1940. Edinburgh: E. & S. Livingstone. 15s. net.

Injuries of the Skull, Brain, and Spinal Cord. Neuro-Psychiatric, Surgical, and Medico-Legal Aspects. Edited by SAMUEL BROCK, M.D., New York University. 9 × 6 in. Pp. 632 + xii, with 63 illustrations. 1940. Baillière, Tindall & Cox. 38s. 6d. net.

Principles of Surgical Care, Shock, and Other Problems. By ALFRED BLALOCK, M.D., Professor of Surgery, Vanderbilt University School of Medicine. 10⅛ × 6⅝ in. Pp. 325, with 13 illustrations. 1940. London: Henry Kimpton. 22s. 6d. net.

Oral Surgery. By STERLING V. MEAD, D.D.S., M.S., Washington, D.C. Second edition. 9⅞ × 6⅜ in. Pp. 1315, with 553 illustrations and 7 coloured plates. 1940. London: Henry Kimpton. 63s. net.

The Periodicity and Cause of Cancer, Leukæmia, and Allied Tumours. With Chapters on Their Treatment. By J. H. DOUGLAS WEBSTER, M.D., F.R.C.P.E., F.F.R., Honorary Director, Meyerstein Institute of Radiotherapy, Middlesex Hospital, London, W.1; Lecturer and Examiner in Radiology, University of London; Examiner in Radiology, Faculty of Radiologists, London; etc. $9\frac{3}{4} \times 6\frac{3}{4}$ in. Pp. 178 + xvi, with 5 plates, 8 charts, and 8 tables. 1940. London: Baillière, Tindall & Cox. 12s. 6d. net.

Histopathology of the Peripheral and Central Nervous Systems. By GEORGE B. HASSIN, M.D., Professor of Neurology, University of Illinois, College of Medicine; Attending Neurologist, Cook County Hospital, Chicago. Second edition. $9\frac{1}{4} \times 6\frac{1}{8}$ in. Pp. 554 + xxv, with 302 illustrations. 1940. London and New York: Paul B. Hoeber, Inc. (Medical Book Department of Harper & Bros.). $7.50.

Field Surgery in Total War. By DOUGLAS W. JOLLY, M.B., CH.B. (N.Z.), Late Major, Spanish Republican Army Medical Service. With a Foreword by Surgeon Rear-Admiral G. Gordon-Taylor, O.B.E., M.A., F.R.C.S., F.R.A.C.S. $7\frac{1}{4} \times 4\frac{7}{8}$ in. Pp. 242 + xiv, with 32 illustrations. 1940. London: Hamish Hamilton Ltd. 10s. 6d.

The Head and Neck in Roentgen Diagnosis. By HENRY K. PANCOAST, M.D., Late Professor of Radiology and Director of the Department of Radiology, University of Pennsylvania; EUGENE P. PENDERGRASS, M.D., Professor of Radiology and Director of the Department of Radiology, University of Pennsylvania; and J. PARSONS SCHAEFFER, M.D., PH.D., Professor of Anatomy and Director of the David Baugh Institute of Anatomy, Jefferson Medical College, $9\frac{7}{8} \times 7$ in. Pp. 976 + xxxii, with 1251 illustrations. 1940. London: Baillière, Tindall & Cox. 69s.

Orthopædic Operations. Indications, Technique, and End Results. By ARTHUR STEINDLER, M.D., F.A.C.S., Professor of Orthopædic Surgery, The State University of Iowa, Iowa City, Iowa. $9\frac{7}{8} \times 6\frac{1}{2}$ in. Pp. 766 + x, with 322 illustrations. 1940. London: Baillière, Tindall & Cox. 50s.

Emergency Surgery. By HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital, London; etc. Fourth edition. $8\frac{7}{8} \times 6\frac{1}{8}$ in. Pp. 944 + xii, with 930 illustrations, a large number in colour. 1940. Bristol: John Wright & Sons Ltd. 63s. net.